TM 8-210

WAR DEPARTMENT

TECHNICAL MANUAL

GUIDES TO THERAPY FOR MEDICAL OFFICERS

March 20, 1942

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MENT,
May 6, 1943.

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TECHNICAL MANUAL

GUIDES TO THERAPY FOR MEDICAL OFFICERS

CHANGES No. 1

WAR DEPARTMENT,
WASHINGTON, May 6, 1943.

TM 8–210, March 20, 1942, is changed as follows:

17. Burns.

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b. Fundamentals of treatment.

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(2) Proper prophylactic measures * * * should be taken. Satisfactory end results, that is, minimal scar formation, largely depend on the avoidance of pyogenic infection; the moist surfaces of burns provide ideal conditions for bacterial growth, and it is therefore of paramount importance to employ, if possible, strict aseptic technic in operating on and dressing burns.

(3) To relieve pain.

c. Treatment.—(1) *General.*—(*a*) Proper steps for the prevention or treatment of shock (par. 15) should be instituted. In the presence of extensive burns, quantities of plasma up to 12 units may be required in the first 24 hours. If available, concentrated normal human serum albumin in appropriate amounts may be used likewise. Transfusion of fresh whole blood is often needed to combat the rapidly developing severe anemia which follows extensive burns; when anemia exists, whole blood transfusion is particularly indicated as a preliminary to skin grafting. Parenteral fluid replacement other than that attained by means of plasma or whole blood transfusion should be accomplished by means of 5 percent glucose in sterile distilled water. The intravenous administration of sodium chloride solution should be reserved for those burn cases in which mineral depletion is pronounced, as when great loss of electrolytes occurs as a result of persistent vomiting.

(*b*) In all cases with moderate to severe burns, prophylactic chemotherapy should be administered. Sulfadiazine is the drug of choice (sulfanilamide may be substituted) with an initial dose of 4.0 grams (60 grains). Subsequent doses of sulfadiazine should be given only under the direction of a medical officer. It should be kept in mind that although sulfonamide therapy may serve to prevent infection, great care must be exercised in employing such therapy in burn cases. The extensive fluid loss and possible kidney damage so common in burn cases increases the danger of renal complications from sulfonamide therapy. Maintenance doses of sulfadiazine should be given in 0.5 gram (7½ grains) doses every 4 hours until such time as adequate kidney function can be demonstrated, under which circumstance the dosage may be increased to 1 gram (15 grains) every 4 hours.

(*c*) Prophylaxis against tetanus (par. 49) is indicated in all patients with second or third degree burns.

(*d*) A prophylactic dose of gas-bacillus antitoxin (par. 48) may be given at the discretion of the medical officer.

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(e) Pain should be relieved by adequate doses of morphine. Pain resulting from an extensive burn can ordinarily be relieved by a dose of ½ grain of morphine. In the presence of pronounced anoxia large doses of morphine are dangerous, and under such circumstances the dose should not exceed ¼ grain.

(2) **Emergency treatment of the burned area.**—(a) The burned surface should be covered with a liberal amount of boric acid ointment, or if this is not available, with vaseline. The burn should then be covered with strips of a sterile fine mesh gauze (44 mesh gauze bandage is satisfactory). Over this should be added a smooth thick layer of sterile gauze dressing (large or small first aid dressings are especially suitable for this purpose). Finally, a gauze or muslin bandage should be firmly applied over the dressings.

(b) Contamination of burned surfaces by organisms from the nose and throat is responsible for most of the more serious infections which subsequently develop. Therefore, to minimize contamination from this source, masking should be practiced by the surgeon and assistants. If masks are not available, mouths should be kept closed.

(c) The prompt administration of plasma, when feasible, constitutes an important element in the emergency treatment of burns.

(3) **Definitive treatment of the burned area.**—The burned area should be treated as follows, standard operating room technic, if possible, being employed, with the patient, as well as all attendants, fully masked:

(a) Ether, benzene, lard, or other detergents should be used to remove grease, if present. The burned area and then, separately, the surrounding skin are to be carefully cleansed, using neutral soap and water; green soap should not be used. Avoid the use of brushes in the cleansing of the burn.

(b) All blisters and loose shreds of epidermis are carefully removed, and this material is saved for bacteriologic study, if feasible. Skin that gives evidence of irreparable damage through its full thickness should be excised (evidence of irreparable damage to deeper layers of skin may not be apparent for several days, and excision in such cases should be done as a secondary procedure). The resulting wound should be handled like any other open surgical wound, primary grafting of skin being carried out, if conditions permit. For painful surgical procedures or dressings, general anesthesia, preferably obtained by intravenous injection, should be employed.

(c) Burns of surfaces of any portion of the body may be treated with boric acid ointment. After thorough surgical cleansing, the burned area should be covered with a generous application of boric acid ointment. Strips of a fine mesh sterile gauze (44-mesh gauze bandage is satisfactory) should be applied. Over this should be added a smooth, thick layer of sterile dressing; this may consist of gauze, absorbent cotton, cotton waste, or cellulose. The dressings should be held in place by an evenly and firmly applied bandage; stockinette or some form of elastic bandage is more effective than the ordinary roller bandage. The dressing should not be disturbed for 10 days unless complications develop. Firm pressure is especially important in the case of burns of the hands and face. Immobilization of the part by splinting should be effected when feasible.

(d) As an alternate method of definitive treatment, burns of all surfaces except those of the hands, face, genitalia, and those involving the circum-

ference of an extremity may be treated with tannic acid and silver nitrate. This method, however, should not be employed in the instance of burns which are grossly contaminated or in which 12 or more hours have elapsed since the receipt of the burn. A freshly prepared 10 percent aqueous solution of tannic acid is sprayed over the burned area. This is followed immediately by spraying the area with a mixture of equal parts of 10 percent tannic acid and 10 percent silver nitrate solutions. This mixture should then be sprayed on the burn every half hour until a satisfactory eschar has been formed. Care should be taken to avoid spraying normal skin about the wound. While drying, the burned area may be kept exposed to the air in a heated cradle. After the eschar is dry, it may be covered by a dry sterile dressing. In the absence of infection, the eschar should be allowed to separate spontaneously. If infection develops, the eschar must be removed from the entire infected area, and the latter should then be treated like any other infected wound, with the employment of appropriate systemic and local therapy (par. 51).

(e) Physiological saline solution has been found useful in the treatment of burns involving the face, hands, and especially fingers, the flexures, and the perineum. It may also be used for the treatment of surface infections following the removal of eschars produced by tannic acid or other agents. Saline may be employed in the form of packs or baths.

[A. G. 062.11 (3-23-43).] (C 1, May 6, 1943.)

24. Care of feet.—*a. Foot hygiene.*

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(4) Excessive moisture plays * * * wear oiled shoes. The Army foot powder is of value. It is composed of salicylic acid 2 percent, desiccated alum 1 percent, starch 10 percent, talcum powder 78 percent, boric acid 6 percent, and zinc stearate 3 percent. The powder is rubbed on the feet and used as a dusting powder for the feet and socks.

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b. Trichophytosis.—(1) Proper cleansing and the control of moisture greatly reduce the incidence of so-called "athlete's foot." Prevention of infection in company bathhouses may be attempted by requiring a foot bath of a few minutes in a solution of calcium hypochloride or sodium hypochloride giving 50 to 100 parts per millim of available chlorine. The Army foot powder is an efficient preventive agent if rubbed on the feet and used as a dusting powder for the feet and socks once or twice daily.

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[A. G. 062.11 (3-23-43).] (C 1, May 6, 1943.)

Section IV is rescinded and the following substituted therefor:

SECTION IV

DIAGNOSIS AND TREATMENT OF VENEREAL DISEASES

36. General.—*a.* This section on the diagnosis and treatment of venereal diseases is based on the opinions and recommendations of the Subcommittee on Venereal Diseases of the Committee on Medicine, Division of Medical Sciences, National Research Council. *Since substantial changes from previous treatment methods are here recommended,* this section should be read carefully by all medical officers who are called upon to treat venereal diseases. It is published as a general guide for medical officers and is to be used with due consideration of factors which may be presented by each individual case. It is not intended that the recommendations contained herein will be used to the exclusion or neglect of other indicated therapeutic or nursing procedures.

b. In the zone of the interior and in the communications zone venereal disease cases will ordinarily be hospitalized for treatment during the infectious stages. In order to maintain effective strength of organization during combat, consideration may be given to the treatment of venereal disease cases on a duty status with their organization in the combat zone.

37. Gonorrhea.—*a. Diagnosis in male.*—(1) A diagnosis of gonorrhea must not be made in the absence of laboratory confirmation. Treatment for gonorrhea should be started at once if the patient has an acute purulent urethral discharge, but material should be obtained before treatment is begun for subsequent laboratory study (smear and/or culture).

(2) *Acute gonorrhea.*—The detection of gram-negative intracellular diplococci in smears of the urethral exudate, or smears of the centrifuged sediment of the first glass of urine, or fluid from conjunctival sac or joints, establishes the diagnosis of gonococcal infection. In competent hands cultures yield a higher percentage of positive results and are of particular value in cases in which no gonococci are found in the smear.

(3) *Chronic gonorrhea* (posterior urethritis, prostatitis, seminal vesiculitis, epididymitis, and arthritis.)—The detection of gram-negative intracellular diplococci in smears of the exudate obtained by digital stripping of the prostate, Cowper's glands, and the urethra, or in smears of the centrifuged sediment of urine passed after stripping the prostate, Cowper's glands, and the urethra, or the demonstration of gonococci in cultures of material so obtained, establishes the diagnosis of gonococcal infection.

b. Diagnosis in female.—(1) A diagnosis of gonorrhea must not be made in the absence of laboratory confirmation. (Treatment for gonorrhea should be started at once in women who have evidence of this disease, even though laboratory studies are negative or not available. If laboratory facilities are not available, material for subsequent laboratory studies should be obtained before treatment is begun.)

(2) The detection of gram-negative intracellular diplococci in smears of material obtained from any of the following: the urethra, Skene's glands, or the cervix (or from Bartholin's glands or the rectum, when clinical symptoms exist); or positive cultures of such material establishes the diagnosis of gonococcal infection. (*Caution:* The normal genital bacterial flora and the flora of nonspecific infections may contain organisms that in smear closely resemble gonococci. Therefore, cultural methods should be utilized when possible.)

c. Gonorrheal ophthalmia.—The diagnosis is made on the basis of an acute purulent conjunctivitis, pus containing gonococci, and rapid involvement of the external coats of the eye. (Sulfonamide therapy should be instituted immediately. Prompt ophthalmological consultation is imperative.)

d. Serologic tests for syphilis.—All patients with gonorrhea should be given a serologic test for syphilis on admission, a test before being discharged to duty, and if possible, a final test 2 months after discharge.

e. Treatment of gonorrhea in males and females.—(1) Local treatment of any kind (injections, irrigations, massages, instrumentations) is contra-indicated in uncomplicated acute gonorrhea.

(2) Treatment should consist of not more than two courses of chemotherapy. A careful inquiry should be made as to previous sulfonamide therapy, recent self-administered chemotherapy for gonorrhea, and for previous drug reactions. (See par. 42.)

(*a*) Sulfathiazole and sulfadiazine are each highly efficient, and in the doses recommended cause toxic manifestations very infrequently. Either of these compounds is recommended. Sulfapyridine, although nearly as efficient as sulfathiazole or sulfadiazine, is more toxic and should be used only if the other compounds are not available. Sulfanilamide is far less effective than the other sulfa compounds in gonorrhea and is not recommended.

(*b*) The recommended dosage for sulfathiazole and sulfadiazine is *1 gram* (*15 grains*) *four times a day for 5 days.* The recommended dosage for sulfapyridine is *1 gram* (*15 grains*) *three times a day for 5 days.*

(*c*) A second course of the drug, in the same dosage, should be given if there is evidence of persistence or recurrence of the disease. There should, however, be a lapse of 5 days between the two courses of medication.

(*d*) Patients in whom the gonococcus is present after the second course, or who have not made a satisfactory clinical response, generally should be transferred to a general hospital.

(*e*) In males, local treatment to the urethra generally should be administered only in a general hospital.

(*f*) In the female, hot vaginal douches (under no more than 2 feet of water pressure) afford comfort and promote cleanliness. Acute pelvic inflammatory disease is an indication for bed rest, ice bags to the abdomen, and analgesics. If enemas are necessary, preliminary bathing of the perineum is indicated before inserting the rectal tube in order to avoid inducing gonococcal proctitis.

f. Treatment of sulfonamide-resistant gonorrhea in males and females (*general hospitals*).—(1) It is recommended that carefully selected patients with sulfonamide-resistant infections be given 10 hours of sustained fever therapy, where such therapy is available, preceded by chemotherapy consisting of sulfathiazole, 1 gram every 6 hours for eight doses. This program should be preceded by 1 week of freedom from sulfonamide therapy. Patients who are not subjected to this form of treatment, and those who are not cured by this means, should be placed upon local treatment.

(2) *Local treatment in male (sulfonamide-resistant cases only).*—(*a*) When the infection is confined to the anterior urethra, an anterior urethral injection, once daily, of not more than 6 cc of a 5 percent solution of mild protein silver or 0.5 percent of strong protein silver is advised. (Retain for 5 minutes.) It should be kept in mind that prolonged use of chemicals tends to perpetuate urethral discharge. Discharge caused by overtreatment of this type may be recognized by the presence of many epithelial cells.

(*b*) All urethral injections are to be administered by a medical officer or a trained attendant; *not by the patient.*

(*c*) Stop all local treatment if the patient develops acute symptoms of posterior urethral infection, such as urgency, painful or marked frequency of urination, or perineal or rectal pain; and confine treatment to hot Sitz baths. When acute symptoms have subsided, resume anterior urethral injections and continue them until prostatic stroking is begun.

(*d*) Extremely gentle prostatic stroking should be tried when the second glass of urine has been clear, and the first glass nearly so, for 2 weeks. If gentle massage is painful or causes a recrudescence of other symptoms, it should not be repeated for 1 week, or until the symptoms have subsided. If it is not painful and if no recrudescence of symptoms occurs, the gland should be gently stripped at 3- and 4-day intervals, and smears of the prostatic secretion examined every 2 weeks. (*When prostatic massage is instituted too soon or applied too vigorously it often induces complications and retards cure.*)

(*e*) Infections of Cowper's glands should be searched for in resistant cases. If these glands are palpable, they should be gently kneaded. This can be accomplished at the time prostatic massage is practiced, by placing the thumb against the perineum and gently massaging first one and then the other gland with the index finger.

(*f*) *No instruments of any type should be passed into the urethra while gonococci are present.*

(3) *Local treatment in female.*—(*a*) See paragraph 37*e* (2) (*f*).

(*b*) The persistence of infection in Skene's glands, Bartholin's glands, or the endocervical glands in spite of the use of measures recommended above constitutes a special problem beyond the province of this directive. Such patients require skilled gynecological treatment.

g. Determination of cure in male (uncomplicated cases).—(1) Cure is determined by the inability to demonstrate gonococci in any of the urogenital fluids including prostatic secretions, by smears or cultures.

(2) Patients whose symptoms have disappeared as the result of sulfonamide medication may have the first test of cure on the third day after disappearance of symptoms. Thus, for example, patients clinically well on the third day of chemotherapy, whose urethral cultures or smears are negative for gonococci, may have on the *sixth day* prostatic massage and may then be discharged to duty if smears of the urogenital fluids are normal. When feasible, these patients should return to the hospital for two subsequent examinations at weekly intervals and should be discharged as cured after three negative examinations.

h. Determination of cure in male (sulfonamide-resistant cases).—(1) Cure is determined by the inability to demonstrate the gonococcus in any of the urogenital fluids by repeated smears or cultures. Material for these studies should be obtained in the manner described in paragraph 37*a* (3). The prostatic secretion

should be included in the study of all types of infection. Three successive negative studies at weekly intervals constitute practical evidence of cure. Tests for cure may be carried out after the patient has returned to duty.

(2) Patients whose symptoms disappear as the result of fever therapy may have tests of cure stated on the second day following the treatment.

i. Determination of cure in the female (uncomplicated cases).—(1) Patients whose symptoms have disappeared as the result of chemotherapy may have tests of cure begun on the third day after disappearance of symptoms.

(2) Cure is determined by—

(*a*) Absence of tender masses or points of tenderness in the pelvis.

(*b*) Inability to demonstrate the gonococcus by smears or cultures in material obtained from the urethra, Skene's glands, Bartholin's glands, or the cervix. Such tests should be repeated every 2 weeks for 3 months, and, if all are found to be negative, the patient should be discharged from observation. These tests should be carried out on an ambulatory basis.

(*c*) To obtain material for smears and cultures, massage the urethra, Bartholin's glands, and Skene's glands, obtaining secretion with small cotton-wrapped applicator or a platinum loop. Pass bivalve vaginal speculum without lubricant, expose cervix, clean vagina and cervical canal, squeeze cervix between ends of speculum blades, and obtain expressed fluid on cotton applicators or platinum loops for smear and culture.

j. Determination of cure in female (sulfonamide-resistant cases).—(1) Patients whose symptoms have disappeared as the result of prolonged artificial fever may have tests of cure begun on the second day after treatment. The tests of cure are the same as those recommended for female patients in uncomplicated gonorrhea.

(2) Patients under local treatment should have smears and culture done at least every 2 weeks. If these tests remain consistently negative for 3 months, and if there are no demonstrable complications, the patient should be discharged from observation.

38. Syphilis.—*a. Diagnosis of syphilis.*—(1) It is of the utmost importance that the diagnosis in early syphilis (primary and secondary stages) be established at the earliest practicable moment and that treatment be instituted as soon as the diagnosis is made.

(2) All ulcerative genital lesions, extragenital lesions characterized by indolence, induration, and regional lymphadenopathy, and cases of urethritis accompanied by indolent enlargement of related lymph glands are to be regarded as probable cases of syphilitic infection until this possibility has been excluded by repeated darkfield examinations and repeated serologic tests. In these cases routine serologic tests will be done not less often than—

(*a*) On admission to sick report.

(*b*) Second week.

(*c*) About the end of the first month.

(*d*) About the end of the second month.

(3) Routine serologic tests will also be made in all cases of gonorrhea at least as often as follows:

(*a*) On admission to sick report.

(*b*) Before return to a duty status.

(*c*) About the end of the second month.

(4) *Antisyphilitic treatment will not be started until the diagnosis of syphilis is definitely established.* The demonstration of the *Treponema pallidum* by darkfield examination is conclusive as to the necessity for the immediate institution of treatment. A persistent completely positive serology even in the absence of confirmatory clinical signs or a history of infection is also diagnostic of syphilis, and usually is an indication for treatment. Positive serologic tests on a single specimen should never be made the basis of treatment in the absence of unmistakable clinical evidence of syphilis. A completely positive precipitation or complement fixation test (Kahn or Wassermann) confirmed by a positive complement fixation test (Wassermann) on a second specimen indicates syphilis provided no negative reactions have been obtained. Acute infection, particularly malaria and possibly vaccination procedures, are believed to give rise at times to transient false positive serologic tests. The presence of these complicating factors should be excluded before making a definite diagnosis of latent syphilis. In the event of incompletely positive or contradictory serologic reactions the test should be repeated until the possibility of technical error is excluded.

b. Treatment of syphilis.—(1) *General principles of treatment.*—(*a*) No treatment is to be given for suspected early syphilis until the diagnosis is made either by darkfield examination or confirmed serologic tests. No therapeutic tests are to be used.

(*b*) Arsenoxide (mapharsen) will be used as the standard arsenical. Neoarsphenamine or other arsenicals *cannot* be substituted in the treatment schedule outlined in table I.

(*c*) If it is necessary to use neoarsphenamine, because of the nonavailability of arsenoxide, the former drug should be given only at weekly intervals in courses of not more than 8 consecutive *weekly* doses.

(*d*) Tryparsamide and fever therapy are not to be used outside of a hospital.

(*e*) Each treatment is to be recorded on the Syphilis Register of the patient or if for any reason a syphilis register is not available, a written record is to be kept and transferred to the standard form as soon as possible. Each entry will include date, drug, dose, and reaction.

(*f*) *It cannot be too strongly emphasized that regularity* of treatment schedule without long or short time variations or lapses is critically important to both infection, control, and cure. Every effort must be made to impress this fact on enlisted men and officers as well as medical personnel.

(*g*) Emphasis should also be placed on the completion by each patient of the *full* schedule of treatment in the time called for regardless of early serologic reversal.

(2) *Treatment of early and latent syphilis.*—(*a*) In view of recent advances in knowledge regarding the toxicity and therapeutic efficiency of arsenoxide, the following recommendations regarding the treatment of early and latent syphilis have been prepared by the Subcommittee on Venereal Diseases of the National Research Council and approved by The Surgeon General.

(*b*) Early (primary and secondary) and latent syphilis of any duration should be treated by an identical treatment system. This treatment may be completed within 26 weeks. (See treatment schedule, table I.)

(*c*) Patients with syphilis, early or latent, should, as a rule, be hospitalized initially to the end that a careful examination may be made and antisyphilitic treatment started. The period of hospitalization ordinarily need not be pro-

longed more than 5 to 7 days. Thereafter, treatment should be continued by unit medical officers; or, in areas where concentration of patients is feasible, in centralized ambulatory clinics established in hospitals. Whenever possible and to minimize loss of time from duty, treatment of nonhospitalized patients should be given after duty hours.

(*d*) At reception centers, vaccinations and other procedures incident to induction may be carried out during the initial period of hospitalization for syphilis, by local arrangements between the commanding officers of the reception center and the station hospital.

(*e*) Treatment schedule for early and latent syphilis is shown in table I.

TABLE I.—*Treatment schedule, early and latent syphilis*

Week		
1		
2		
3		Bismuth subsalicylate intramuscularly once weekly, 5 doses.
4	Arsenoxide (mapharsen) intravenously twice weekly, total 20 injections.	
5		
6		
7		
8		*Omit* bismuth—5 weeks.
9		
10		
11		
12		
13	*Omit* arsenoxide (mapharsen)—6 weeks.	Bismuth subsalicylate—intramuscularly once weekly—6 doses.
14		
15		
16		
17		
18		
19		Omit bismuth—5 weeks.
20	Arsenoxide (mapharsen) as in first course, twice weekly total 20 injections.	
21		
22		
23		
24		Bismuth subsalicylate intramuscularly once weekly, 5 doses.
25		
26		

Arsenoxide (mapharsen) dosage: Adjusted approximately to body weight; average dose 60 milligrams, minimum dose 50 milligrams, maximum 70 milligrams.

Bismuth subsalicylate in oil dosage: The standard dose is 0.2 gram of bismuth subsalicylate intramuscularly (not 0.2 gram of elemental bismuth metal).

1. It is anticipated that individuals now on antisyphilitic therapy in accordance with other schedules will continue their antisyphilitic therapy in accordance with the provisions of table I as soon as the requisite drugs are obtained.
2. In effecting this change of schedule it should be borne in mind that the purpose of the new plan of treatment is to furnish adequate

antisyphilitic therapy in a shortened period of time. The drug of paramount importance is the arsenical. Therefore, the technique of change to the new schedule should provide, wherever practicable, that the amount of arsenical drug administered under a previous schedule, together with that administered under the schedule of table I, should equal the total number of doses (i. e., 40) of arsenical drug advocated in the new treatment schedule of table I, and a minimum of 16 doses of bismuth subsalicylate. In transferring from one schedule of treatment to the other it will ordinarily suffice to base the transfer solely on the number of arsenical treatments the individual has received. For example, if a patient has received 17 doses of arsenical drug under a previous schedule, he should be started on the eighteenth dose of mapharsen on the schedule of table I. This method of transfer should be modified if it would at any time call for a patient receiving more than 25 consecutive doses of an arsenical drug. The treatment schedule should always be concluded with an overlapping series of 5 weekly injections of bismuth subsalicylate, as suggested in table I.

3. This plan of treatment is not to be construed to mean, however, that an individual who has completed the arsenical treatments under a previous plan of therapy need be given additional arsenical in order that the total arsenical administered may equal that suggested in table I.

4. In the case of an individual who has been recently inducted into the Army and who had received antisyphilitic treatment prior to entrance, the principles outlined above may be followed, using the physician's or clinic's record of his previous treatment as a basis for further therapy. Such records will ordinarily be readily available upon the surgeon's written request, accompanied by the individual's written authorization for furnishing the data desired. (This paragraph is included in view of the contemplated induction of individuals who have early or latent syphilis without disabling lesions, when facilities for their housing and treatment have been provided.)

5. Patients with cerebrospinal, cardiovascular, or other types of visceral syphilis require specialized supervision and prolonged hospital care. Such cases should be discharged on CDD if discovered soon after induction. Otherwise, they should be transferred to a general hospital for treatment and disposition.

(3) *Technical suggestions.*—(*a*) Discard discolored drugs and solutions and damaged ampules.

(*b*) Dissolve arsenoxide in sterile distilled water in the proportion of 10 milligrams of drug per 2 cc of water; a dose of 60 milligrams will then be contained in 12 cc of solution, 50 milligrams in 10 cc, and 70 milligrams in 14 cc.

(*c*) Shake and aerate arsenoxide; do not shake or aerate the other arsenicals.

(*d*) Inject arsenoxide rapidly to avoid thrombosis; there is little danger of speed shock or nitritoid crisis. Other arsenicals should be injected *slowly* to avoid speed shock or nitritoid crisis.

(*e*) Thoroughly shake oily suspensions.

(*f*) Attempt aspiration after insertion of needle before making any injection, especially intramuscularly.

(*g*) Inject bismuth intramuscularly into upper outer quadrant of buttock. Alternate sides.

(*h*) Massage firmly after withdrawing needle from buttock and have patient prolong massage to 3 minutes.

(*i*) Advise rest, if practicable, after arsenicals.

(*j*) Warn patient to report his reactions.

(*k*) Watch mouth and gums for bismuth stomatitis.

(4) Treatment is to be stopped and the patient hospitalized if the following appear:

(*a*) An itchy dermatitis of the face or flexures.

(*b*) Jaundice.

(*c*) Petechial or other hemorrhagic lesions.

(*d*) Evidence of cerebral injury, even though slight.

(5) *General antireaction therapy.*—(*a*) Epinephrin solution 1:1000, ½ to 1 cc subcutaneously for speed shock or nitritoid crisis.

(*b*) Glucose 500 cc 5 percent solution intravenously supplemented with thiamine chloride 5 milligrams, for jaundice.

(*c*) Vitamin B complex is recommended in suspected liver damage.

(*d*) In cerebral vascular accidents, measures to be considered are venesection, and hypertonic saline solution intravenously (500 cc of a 1.5 percent solution).

(*e*) In blood dyscrasias, transfusions.

(*f*) Sodium thiosulphate for any type of treatment reaction is considered valueless.

(6) *Serologic control of treatment.*—In patients with early syphilis a serologic test will be done at the beginning and end of the schedule of treatment outlined in table I; but treatment may be stopped whether the serologic test for syphilis (STS) is positive or negative. After the completion of treatment, the STS should be repeated 3 and 6 months later. If the test is negative after 6 months, the case may be classified as "Result satisfactory" and the Syphilis Register may be closed. If the test is positive after 6 months, the patient should be referred to a station or general hospital. In patients with latent syphilis the STS should be repeated at the completion of treatment outlined in table I, but the Syphilis Register may be closed when this treatment is completed, regardless of the result of serologic test.

(7) *Spinal fluid examination.*—Should be performed in a hospital in patients with early syphilis at the end of the course of treatment outlined in table I, or as soon as possible thereafter; but in any event before the Syphilis Register is closed. In apparent latent syphilis, spinal puncture should be performed in a hospital before treatment or as soon as possible thereafter, but in any event before the Syphilis Register is closed.

(8) *Control of relapse and infectiousness.*—(*a*) Early syphilis is to be regarded as infectious until the second injection of arsenoxide has been given.

(*b*) Physical inspection of skin (including palms and soles), mucosae, anus, and genitalia should be performed as often as circumstances permit during treatment and at each probationary inspection.

(*c*) The involution of the chancre or secondaries should be watched to detect treatment-resistant cases.

(*d*) Patients should be warned to look for and to report mouth, skin, and genital lesions. Darkfield examination is of great help in recognizing relapsing lesions of the skin, mucosa, and genitalia.

(9) *Complications or relapse.*—In the event of any complication of treatment (serious treatment reactions) or any evidence of relapse, clinical or serologic, the patient should be at once transferred to a station or general hospital.

(10) Cardiovascular, visceral, and neurosyphilis require special treatment in hospital. For additional details see standard texts.

(11) *Treatment of precocious late syphilis (tertiarism).*—As soon as possible, precocious late syphilis (early gummatous and rupial lesions and bone lesions) should be hospitalized for combined fever and arsenical therapy.

(12) *Treatment of congenital syphilis.*—On recognition or on appearance of active lesions, congenital syphilis should be treated on the schedule for early and latent syphilis.

(13) *Separation from the service.*—When practicable, the physical status of every patient with syphilis, whether the disease was acquired before or after induction into the service, will be completely reevaluated when discharge from the Army is contemplated.

c. Diagnostic nomenclature for syphilis in Army.

Syphilis, primary.
Syphilis, secondary.
Syphilis, early latent (less than 4 years since infection).
    Spinal fluid negative.
    Spinal fluid not performed (diagnosis tentative).
Syphilis, late latent (4 or more years since infection).
    Spinal fluid negative.
    Spinal fluid not performed (diagnosis tentative).
Syphilis, tertiary—otherwise unclassified.
    Mucocutaneous.
    Osseous.
    Ocular (except optic atrophy).
    Visceral (except cardiovascular).
    Cardiovascular—other.
        Aneurysm (saccular).
        Aortic regurgitation (insufficiency).
        Aortitis (uncomplicated).
    Neurosyphilis—otherwise unclassified.
        Asymptomatic.
        Acute syphilitic meningitis.
        Diffuse meningovascular.
        Optic atrophy.
        Tabes dorsalis.
        Taboparesis.
        Psychosis with syphilitic meningoencephalitis (general paresis).
        Psychosis with neurosyphilis other than paresis or taboparesis.
Syphilis, type undetermined—to include cases in which accurate diagnosis has not been made.
Syphilis, congenital—include all manifestations.
Arsenical poisoning.
Bismuth poisoning.
Iodine poisoning.
Mercury poisoning.
Spinal puncture for diagnosis or progress.

Special therapeutic practices:
Fever therapy—
Malaria.
Artificial.

d. Definition of terms and explanations of their use.—(1) *Primary.*—To include those cases presenting the primary lesion of syphilis (the chancre), which have not yet developed secondary manifestation. This diagnosis must be confirmed by darkfield examination, serologic test of the blood, or both. If blood serologic test is negative, the diagnosis of primary syphilis is not permissible without the demonstration of *T. pallidum* by darkfield.

(2) *Secondary.*—To include only those cases of early syphilis which show one or more of the manifestations of systemic dissemination of the spirochete; for example, generalized enlargement of lymph glands, cutaneous eruption, mucous patches, condylomata lata, patchy alopecia, laryngitis, bone pains, febrile reaction, and so forth. The chancre may or may not be present, and if present, may be in any stage of evolution. In early secondary cases the manifestations of systemic spirochetal dissemination are increasing, have attained their maximum, or are waning. This diagnosis must be confirmed by darkfield examination, serologic test, or both. In early secondary syphilis and in addition to the manifestations listed above, ocular or neurologic complications (iritis, neuroretinitis, acute syphilitic meningitis) should be specially recorded as: "Syphilis, secondary, manifested by ——————."

(3) *Latent.*—Secondary symptoms have subsided and the active manifestations of late syphilis have not yet supervened. There are no evidences of syphilis other than a positive serologic test of the blood. Cases will be classified as: "Latent (early or late)—spinal fluid negative" or "Latent (early or late)—spinal fluid not performed (diagnosis tentative)." The date of the negative examination of the spinal fluid will be stated in all cases.

(4) *Tertiary.*—This classification is to be limited to cases that show active lesions of late syphilis. The lesion may be a gumma or it may be a diffuse process and may involve any organ or tissue of the body. The majority of all patients with tertiary syphilis will fall within six categories:

(*a*) *Mucocutaneous.*—Late syphilitic gummatous lesions of skin or mucous membrane.

(*b*) *Osseous.*—Periostitis, osteomyelitis, arthritis, and synovitis.

(*c*) *Ocular.*—Iritis, uveitis, keratoiritis, keratitis, and choroiditis, but not including optic atrophy.

(*d*) *Visceral.*—Hepatic, gastric, etc., but not including cardiovascular.

(*e*) *Cardiovascular.*—To include all lesions of the heart and great vessels. Classify as follows:

1. *Aneurysm* (saccular).—Do not use for a fusiform dilation of the aorta. Specify artery involved.
2. *Aortic regurgitation* (*aortic insufficiency*).—Specify whether with or without cardiac decompensation.
3. *Aortitis, uncomplicated.*—To be used only for those patients with symptoms and physical or X-ray signs of syphilitic aortic involvement in the absence of aneurysmal sacculation or aortic regurgitation.

(5) *Neurosyphilis.*—To include all cases with involvement of the central nervous system, classified as follows.

(*a*) *Asymptomatic.*—To be used only for those patients with early or late syphilis who have no symptoms or detectable physical signs of central nervous system involvement, and in whom the diagnosis is based on the routine finding of abnormalities in the spinal fluid.

(*b*) *Acute syphilitic meningitis.*—Usually occurs within the first 2 years of the disease, most commonly as a relapse phenomenon (neurorecurrence), manifested by the usual signs of low grade meningeal involvement, with or without cranial nerve palsies.

(*c*) *Diffuse meningovascular.*—This is a catch basket category to include all patients with neurosyphilis who do not fit into other diagnostic categories enumerated. Manifestations to be stated in each instance.

(*d*) *Optic atrophy.*—Primary or secondary.

(*e*) *Tabes dorsalis.*—Manifestations to be stated.

(*f*) *Taboparesis.*—To be used only in patients with definite psychiatric signs of paresis complicated by definite clinically demonstrable evidence of damage to the posterior columns of the spinal cord.

(*g*) *Psychosis with syphilitic meningo-encephalitis (general paresis).*—To be limited to cases which show psychic changes in addition to neurologic signs and the characteristic changes in the spinal fluid. Cases with paretic type spinal fluid but without psychic changes will be reported as: "Syphilis, diffuse meningovascular, manifested by_____."

(*h*) *Psychosis with neurosyphilis.*—To include neurosyphilis with psychosis other than cases of paresis and taboparesis.

(*i*) *Unclassified.*—If tertiary manifestations occur which do not fit into one of these categories, diagnose as: "Syphilis, tertiary, otherwise unclassified"—Specify.

(6) *Type undetermined.*—To include cases in which accurate diagnosis has not been made. Every effort should be made to make a complete examination and proper diagnostic classification in all cases.

(7) *Congenital.*—To be limited to cases that show definite evidence of the existence or former existence of the characteristic changes of congenital syphilis, such as interstitial keratitis, Hutchinson's teeth, saber shins and other bone changes, saddle nose, eighth nerve deafness, and so forth. The congenital origin of syphilis is not to be assumed merely because the time and the circumstances of the infection cannot be ascertained and there is no scar of a primary lesion.

(8) *Drug poisonings.*—The suggested terms for various drug poisonings are intelligible as they stand.

(9) *Spinal puncture for diagnosis or progress.*—This should be used in all cases (syphilitic or otherwise) routinely hospitalized for purpose of a spinal puncture.

(10) *Special therapeutic practices.*—This diagnostic grouping has been included for it seemed desirable to indicate special therapeutic practices which necessitate hospitalization.

39. Chancroidal infection.—*a. Definition.*—Chancroid is a venereal disease transmitted only by direct contact and characterized by single or multiple genital ulcers. The latter possess irregular crater-form margins, are usually not indurated, and exhibit a tendency toward the formation of complicating suppurating inguinal adenitis. The incubation period is usually 3 to 14 days.

b. Diagnosis.—It is important to rule out the presence of mixed syphilitic and chancroidal infection. For this purpose at least 3 darkfield examinations should be made on successive days; a blood serologic test for syphilis should be made

on admission to the hospital, during the second week, and at monthly intervals for 2 months following healing of the chancroidal lesions. Laboratory tests for the diagnosis of chancroid (Ito-Reenstierna skin test or the staining or cultural isolation of the Ducrey bacillus) are not recommended.

c. Treatment.—(1) *Chemotherapy.*—(*a*) *Local.*—Accessible lesions should be cleaned with soap and water and dried. They should then be completely covered with powdered sulfanilamide and a loose, dry dressing applied. This should be repeated at daily intervals until the lesion heals. Other local medication is not recommended. In patients with tight phimosis and underlying ulcerative lesions, the phimotic preputial cavity should be irrigated twice daily with 1–5000 potassium permanganate solution.

(*b*) *Systemic.*—Administer sulfathiazole or sulfadiazine 1 gram (15 grains) 4 times a day for 5 days. Sulfanilamide 1 gram (15 grains) 3 times a day for 5 days may be utilized instead of sulfathiazole or sulfadiazine, but is less well tolerated. Practically all chancroidal infections will respond to the above routine. In fact, if the lesion does not heal, doubt is cast on the correctness of the diagnosis of chancroid, and the patient should be restudied from the diagnostic standpoint, and, if necessary, treated surgically.

(2) *Surgical therapy.*—(*a*) Surgical procedure designed to relieve phimosis or paraphimosis should be resorted to only on the basis of sound clinical judgment.

(*b*) *Chancroidal bubo.*—Most of these will subside with systemic sulfonamide therapy. If extensive suppuration is present, the bubo may be opened by a small incision, the pus aspirated, and the cavity packed with sulfanilamide powder.

40. Lymphogranuloma venereum.—*a. Definition.*—This disease concept includes the conditions formerly known as lymphogranuloma inguinale, lymphopathia venereum, climatic bubo, esthiomene, and inflammatory rectal stricture.

b. Etiology.—A filtrable virus, probably multiple strains.

c. Geographic distribution.—World-wide.

d. Clinical picture.—A systemic disease of the lymphatic system, usually originating in a trivial and transitory lesion of the penis, vulva, vagina, or rectum, which frequently escapes the patient's notice. The invasion of the lymphatic glands usually occurs from 10 to 30 days after infection, occasionally is delayed months. Inguinal adenitis is often bilateral and occasionally subsides without suppuration. During this stage, constitutional symptoms may be observed. Lymph nodes may fuse to skin, resulting in multiple areas of softening, followed by numerous fistulae. Extensive scarring accompanies healing. The anorectal syndrome usually is found only in the female, and is characterized by rectal pain, discharge of blood and pus from the anus, a tendency toward extreme chronicity, and the production of rectal stricture.

e. Differential diagnosis.—Differentiate from malignant tumors, Hodgkin's disease, tularemia, tuberculosis, pyogenic infections, chancroidal bubo, and syphilis. Mixed venereal infections should be ruled out by the darkfield examination of material from genital lesions for the causative organism of syphilis. Frequent serologic examinations should be continued for at least 2 months after the disappearance of the lymphatic symptoms.

f. Laboratory tests in lymphogranuloma venereum.—(1) Only one diagnostic procedure for lymphogranuloma venereum, the intradermal test of Frei, has as yet come into general use. Other methods used in confirming the diagnosis are either impractical (animal inoculation, artificial cultivation of the virus, non-

specific (alterations in serum protein), or their value not yet established (complement fixation).

(2) *Frei antigens.*—(*a*) *Chick embryo antigen.*—This is the preparation recommended for Frei testing. Nonspecific reactions occur, so that simultaneous inoculation of control material must always be made.

(*b*) *Human bubo-pus antigen.*—Pus is obtained from an unruptured bubo from an acute case of lymphogranuloma venereum. This pus must be diluted 1:5 or 1:10, cultured for sterility, and heated for 1 to 2 hours on consecutive days at 58° C., following which a preservative is added. This material is placed in sterile rubber stoppered vials and is then tested upon known positive cases and known negative controls to determine its potency. Pus from different patients differs in antigenic potency. When human bubo-pus antigens are locally available they may be used, but are not generally recommended for use by the armed forces because of the uncertainty of sources of supply and the variability of antigens.

(*c*) *Mouse brain antigen.*—This preparation is commercially available. However, it is not recommended for use because it yields a high proportion of nonspecific results.

(3) *Frei test.*—(*a*) *Method of use of chick-embryo antigen.*—This preparation is supplied in two ampules, one of which is the virus-containing antigen, the other the nonvirus-containing yolk sac control. For use in Frei testing, 0.1 cc of antigen and 0.1 cc of control material are injected *intradermally* into different areas on the flexor surface of the forearm. The areas chosen should be at least 4 centimeters apart, or the virus antigen and control may be injected in opposite forearms. Separate tuberculin syringes and 26-gage needles should be used.

(*b*) *Reading of results.*—The injected areas must be inspected 48 and 72 hours later. A positive reaction consists in a more or less indurated papule 7 millimeters or more in diameter (disregarding the surrounding zone of erythema), with or without central vesiculation or ulceration. *A doubtful reaction* consists in a papule roughly from 5 to 7 millimeters in diameter, without central ulceration or vesiculation. A negative reaction consists in no change at the injected site, or erythema only, or a papule less than 5 millimeters in diameter. If the control material likewise yields a positive papule (7 millimeters or over), the Frei test should be repeated with human bubo-pus antigen if it is available. The *test* may be read as positive or doubtful if the papular reaction described occurs only with the virus-containing antigen, the control remaining negative.

(4) *Interpretation of results of Frei test.*—The Frei test is of greatest value in patients with the acute bubonic form of lymphogranuloma venereum where a negative test may be observed to develop gradually into a positive one. There is reason to believe that in certain instances there are nonspecific cross-reactions in the presence of other venereal diseases, for example, chancroid, granuloma inguinale, syphilis; and a positive Frei test with any antigen must be interpreted with caution when suspected lymphogranuloma venereum coexists with these diseases. Moreover, a positive Frei test cannot be relied upon absolutely to establish the lymphogranulomatous nature of any clinical condition, since it is known that, in untreated infections with lymphogranuloma venereum, skin sensitivity persists for many years, probably for a lifetime. A positive test may therefore mean only that the patient has had lymphogranuloma venereum at some time in the past, rather than that his present symptoms are caused by this disease. In short, *the Frei test is of most diagnostic value when it is negative,* since under these circumstances lymphogranuloma venereum, past or present, may be excluded with reasonable certainty. *A diagnosis of lymphogranuloma venereum*

should not be made on the basis of a positive Frei test in the absence of clinical signs.

g. Treatment.—(1) *Local.*—Patients with acute inguinal adenitis should be hospitalized whenever possible. The fluctuant nodes may be aspirated, but incision and drainage should be delayed until the effect of chemotherapy has been observed. Radical excision is inadvisable because of the risk of elephantiasis of the scrotum or vulva.

(2) *Chemotherapy.*—(*a*) The value of the sulfonamide compounds in lymphogranuloma venereum has not been definitely established, but preliminary reports indicate that they may be effective. Sulfathiazole and sulfadiazine are probably the drugs of choice, although sulfanilamide may be used.

1. Sulfathiazole or sulfadiazine should be administered in doses of 1 gram (15 grains) four times daily for 5 days. It may be necessary to prolong this medication to 10 to 14 days, in which case the dose should be reduced to 0.5 grams four times a day.
2. Sulfanilamide, if used, should be administered in doses of 1 gram (15 grains) three times a day for 5 days, followed by a reduction to 0.5 to 0.75 grams three times daily for an additional 5 to 7 days.

(*b*) The acute anorectal syndrome should be treated in the same manner as the inguinal manifestations. Stricture or other late complications should receive special consideration.

41. Granuloma inguinale.—*a. Definition.*—Granuloma inguinale is a chronic disease due to infection with a leishmania-like organism. It involves primarily skin and mucous membranes, rarely with coincident adenopathy; is characterized by vivid-hued, shining verrucous, vegetating nodules of granulating tissue with a hemorrhagic surface surrounded by a thin, easily excoriated epidermis. The condition spreads by peripheral extension and antoinfection, often involving the entire genital area. It may involve large adjacent areas of the lower abdomen and thighs. The lesions show little or no tendency to spontaneous healing and may persist for months or years.

b. Diagnosis.—The clinical appearance of a chronic process involving the groin and genital areas with little involvement of the lymph nodes is characteristic of the disease. The finding of Donovan bodies by Wright stain in deep tissue scrapings or biopsy of a peripheral area of diseased tissue (including a section of normal adjacent skin) confirms the diagnosis. Lymphogranuloma venereum, chancroid infection, and syphilis should be considered in the differential diagnosis, and appropriate diagnostic tests should be performed.

c. Treatment.—(1) Tartar emetic administered intravenously in doses of 0.03 to 0.12 grams. Patient should remain recumbent for 1 hour following an injection. Drug intolerance is indicated by nausea, vomiting, cough tachycardia, and hypotension. Initiate treatment with 3 cc of a freshly prepared 1-percent solution. Each subsequent dose, freshly prepared, should be increased 3 cc if tolerated until the aximum dose, 12 cc, is attained. The maximum tolerated dose is given three times weekly for 15 treatments.

(2) Fuadin 1 to 3 cc (0.06 to 0.18 grams) or *anthiomaline* 1 to 3 cc (0.06 to 0.18 grams) (both of these are complex antimony compounds) may be given intramuscularly two or three times weekly for 20 to 25 doses when the patient has difficulty in taking tartar emetic, or when the lesions have not improved satisfactorily under the former drug.

(3) Courses of tartar emetic, fuadin, or anthiomaline, separated by "rest periods" of 2 weeks, should be continued for at least 4 months after all lesions are completely healed, otherwise relapse is almost certain to occur.

(4) Local treatment of the lesions may be limited to daily dressings; or surgical excision of the entire area may be necessary. Large areas may be treated with solid carbon dioxide pencils. Deep X-ray therapy in expert hands has yielded promising results.

[A. G. 062.11 (3–23–43).] (C 1, May 6, 1943.)

42. General.

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(7) *Laboratory procedures used in following patients receiving sulfonamide compounds.*—(*a*) *General.*—It should be constantly borne in mind that laboratory measures should be utilized when feasible as an adjunct in the effort to avoid complications from the administration of sulfonamide compounds. Careful clinical observation, hemoglobin estimation, and red and white blood counts should be carried out; nevertheless, in all cases, and in those showing any abnormal response to the drug or having a history of such an abnormal response, and in those who have recently taken sulfonamides, or who are suspected of having done so, action of the drug should be carefully followed by appropriate additional laboratory procedures. Hospitalization should be promptly effected in case of complications.

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[A. G. 062.11 (3–23–43).] (C 1, May 6, 1943.)

51. Wound infections.—*a. General.*—The problem of * * * certain specific agents. The following statements refer to the definitive treatment of wounds and are not to be confused with first aid and emergency measures applicable to the handling of battle casualties.

b. Prophylaxis.

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(4) Chemotherapy should be instituted immediately after debridement of the wound.

(a) Crystalline sulfanilamide should be lightly dusted on the wound.

(b) Give 2.0 grams (30 grains) sulfadiazine by mouth, and then 1 gram (15 grains) every 6 hours, day and night, for 7 days or until danger of infection has passed, or there are indications for a change of treatment. The amount of a sulfonamide already taken by the injured man should be ascertained and the subsequent dosage reduced accordingly. In most instances the wounded man will have received one 4-gram dose of sulfadiazine orally and will have had crystalline sulfanilamide applied locally to the wound prior to debridement. Proper consideration to dosage should also be given to patients with urinary suppression due to hemorrhage, shock, or dehydration. If adequate amounts of fluid (2,500 to 3,000 cc) cannot be given, the dosage also should be lowered, to prevent an abnormally high concentration of the drug.

(c) In cases in which sulfadiazine cannot be given by mouth it can be given entravenously (par. 43).

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[A. G. 062.11 (3–23–43).] (C 1, May 6, 1943.)

Section VI is rescinded and the following substituted therefor:

SECTION VI

TREATMENT AND CONTROL OF CERTAIN TROPICAL DISEASES

54. General.—The following notes on the treatment and prevention of certain tropical diseases are based on recommendations of the Subcommittee on Tropical Diseases and the Committee on Medicine of the Division of Medical Sciences, National Research Council, and were adopted after consultation with representatives of the National Institute of Health and the Naval Medical School, Bethesda, Maryland. The information is offered only as a general guide in the handling of tropical diseases and is for use at the discretion of the medical officer. It is not intended as a substitute for the more comprehensive publications available on the subject.

55. Cholera.—*a. Etiologic agent.—Vibrio comma.*

b. Geographic distribution.—The disease is endemic in Asia. Since ancient times many pandemics have originated in endemic centers in India, and have spread over both tropical and temperate regions of the world.

c. Transmission.—Through the ingestion of food or drink contaminated with feces containing *V. comma.* Flies are important vectors. Few patients remain carriers of the vibrios for more than 10 days, but in epidemic areas there is also a small percentage of healthy contact carriers who may excrete vibrios for a month or more.

d. Recognition.—Incubation usually 2 to 3 days. Onset usually sudden, with profuse, painless "rice water" diarrhea and vomiting, shrinking of face and soft tissues due to loss of body fluids, muscular cramps, suppression of urin, and severe prostration. There are mild ambulatory cases and there is a fulminant type (rare) in which death may occur before purging has begun.

e. Specific diagnosis.—By identification of *V. comma* in cultures of feces. Fecal smears stained with carbol fuchsin diluted 1 to 10, showing the comma forms with "fish in stream" appearance, are highly suggestive.

f. Treatment.—(1) *Restoration of body fluids.*—This is the most important therapeutic objective and should be promptly and adequately attacked. Fluids should be given liberally by mouth unless contra-indicated by vomiting or nausea. It will usually be necessary to supplement oral administration by parenteral injections. This may be accomplished by—

(*a*) The intravenous administration of hypertonic saline solution prepared as follows:

Sodium chloride	13.75 grams.
Calcium chloride	0.25 gram.
Distilled water	1,000.00 cc.

This aids in replacing salt lost by diarrhea and assists in retaining fluid in the blood vessels, thus maintaining the blood pressure and increasing the excretion of urine. The average cholera patient will require 2 liters of this solution every 6 to 8 hours for 1 or 2 days. The injection should be given slowly and continuously and it may be advisable to tie in a canula because of extreme restlessness or of collapsed veins. The pulse and blood pressure should be watched carefully and if there is no suitable response to a given injection it should be repeated within 2 to 3 hours.

(*b*) The intravenous or subcutaneous administration of normal saline, 1,000 cc every 4 hours until dehydration is relieved. In giving large amounts of parenteral fluid, caution should be taken not to exceed the requirements for normal hydration. If time and equipment permit, specific gravity of the blood may be used as a guide to fluid requirements. This may be determined as follows:

1. Prepare a series of solutions of glycerin and distilled water, of specific gravities 0.002 apart, from 1.050 to 1.070 (i. e., 1.050, 1.052, 1.054, etc.). Place small portions (10 to 15 cc) of these solutions in small bottles. Place one drop of blood in each bottle. The specific gravity of the blood is indicated by the bottle in which the drop of blood neither rises to the top nor sinks to the bottom of the solution.

2. Administer the saline slowly and continuously, the amount to depend on the specific gravity of the blood as follows:

If the specific gravity is 1.062, give 1,000 cc.
If the specific gravity is 1.063, give 1,500 cc.
If the specific gravity is 1.064, give 2,000 cc.
If the specific gravity is 1.065, give 2,500 cc.

Repeat saline injections every 4 hours until specific gravity of blood drops below 1.062. The normal is 1.056 to 1.058. If the patient is dehydrated and equipment for determining specific gravity of the blood is not available, administer hypertonic or normal saline, using judgment as to amount.

(2) *Treatment of acidosis and suppression of urine.*—To combat anuria or marked acidosis, use the following solution intravenously:

Sodium chloride	5. 75 grams.
Sodium bicarbonate	18. 25 grams.
Distilled water	1, 000. 00 cc.

This solution should not be sterilized by boiling or autoclaving, as the temperatures reached during these procedures may change the bicarbonate to the caustic carbonate. The following technique may be employed: Dissolve the sodium chloride (5.75 grams) in the distilled water (1,000 cc) and sterilize by boiling. Remove from the heater and at once add sodium bicarbonate (18.25 grams) which has been taken directly from the original container and weighed in a sterile vessel. The solution should be cooled to body temperature and used at once. This solution should be prepared and administered with great care and the *patient observed carefully for signs of tetany or other manifestations of alkalosis.*

(3) *Control of shock.*—(*a*) In stage of collapse, add 50 grams of glucose to each 1,000 cc of saline solution administered, injecting not more than 1,000 cc per 30 minutes or more than 400 grams of glucose daily. If sugar appears in the urine, insulin may be given hypodermically as indicated. Glucose solutions for intravenous injections should be supplemented with 2 milligrams thiamine hydrochloride for each 50 grams of glucose. If normal human serum or plasma is available for intravenous use, it may be used as a means of controlling shock, but not as a substitute for other fluids which are essential.

(*b*) Keep the patient in bed and apply heat to the abdomen and extremities as long as required. Watch the blood pressure, and if below 100 systolic, give saline or plasma as indicated above.

(4) *Oral medication.*—The use of permanganate of potash and kaolin has been advocated and may be tried when practicable.

(5) *Diet.*—When patient can tolerate food, the diet should be low in residue and supplemented by multivitamin capsules.

g. Prevention.—(1) It is of the utmost importance to protect food and water supplies and to sterilize the excreta of cholera patients. The usual measures for the prevention of enteric infections should be intensified in the presence of cholera, especially as regards flies and quarantine. Only boiled or chlorinated drinking water should be used. Uncooked salads, unpeelable fresh fruits, and raw shellfish should be avoided. (See AR 40–205 and 40–210.)

(2) Troops entering endemic areas should be immunized with cholera vaccine as prescribed by The Surgeon General. Immunity is of short duration, probably not more than a few months. Initial immunization consists of two doses of

cholera vaccine given subcutaneously with an interval of 7 to 10 days between injections. The first dose is 0.5 cc, the second dose 1.0 cc. A stimulating dose of 1 cc of vaccine should be administered every 4 to 6 months as long as danger of infection is present. (See sec. VII.)

56. Dengue.—*a. Etiologic agent.*—The dengue virus.

b. Geographic distribution.—May occur in sudden epidemics of widespread proportions in almost any part of the tropical or subtropical world, wherever the vector, the *Aedes* mosquito, is found. Common in the West Indies, the Pacific Islands, and in the countries about the China Sea and the Mediterranean.

c. Transmission.—*Aedes aegypti* is the principal vector; also *A. albopictus* (Philippines), and possibly *A. taeniorrhynchus* (Florida). The patient is infective to the mosquito from a few hours before onset until the third or fourth day of the disease. Infected mosquitoes become infective on the eighth to eleventh day and remain infective for life.

d. Recognition.—After an incubation period lasting from 7 to 9 days there is sudden onset of fever, "saddleback" in type, ranging from 102° F. to 105° F. The pulse is slow in relation to the fever. There is intense postorbital aching with sharp pains on eye movements. Extremely severe pains in joints and muscles give this disease its name of "break-bone" fever. Leukopenia is present. Commencing on the third to the fifth day, the symptoms abate. Two or 3 days later there is a recurrence of fever and pain accompanied by a rash resembling the eruption of measles or scarlet fever, beginning on hands and feet and spreading to other parts. This secondary fever lasts from 1 to 2 days. Different epidemics show great variations in the clinical picture of the disease. The secondary fever and rash may be lacking. Lymph node enlargement is occasionally a prominent symptom.

e. Specific diagnosis.—None.

f. Treatment.—Symptomatic.

g. Prevention.—Screening, bed nets, spray killing of mosquitoes, and other anti-*Aedes* measures should be energetically prosecuted (see par. 71.11).

57. Dengue-like fevers.—There are several dengue-like febrile diseases in various parts of the world, and some of these may be of importance to military personnel.

a. Sandfly fever (Pappataci fever, Phlebotomus fever, 3-day fever).—This disease is prevalent in the countries bordering the Mediterranean and in China, India, and East Africa. Recently it has been reported in northern Argentina. It is caused by a virus and is transmitted by sandflies (*Phlebotomus*). *P. papatasii* is the principal vector; others are *P. minutes* and *P. perniciosus.* Clinically the disease resembles dengue except for the short duration (2 to 3 days) and, commonly, the absence of a recurrent phase and of the rash. There is no specific diagnosis. The treatment is symptomatic. Prevention is based chiefly on measures against sandflies. Screening with the ordinary mosquito net does not bar the small sandflies but when nets are sprayed with pyrethrum insecticides the sandflies are repelled. Sandflies being poor flyers, breezes from electric fans may prevent biting; upper floors provide some security.

b. Panama 6-day fever.—In the Canal Zone.

c. Van der Scheer 5-day fever.—In the Dutch East Indies.

d. Seven-day fever.—In India. Not to be confused with 7-day fever caused by *L. hebdomadis.*

e. Bwamba fever.—In Uganda, Africa.

f. There are other similar fevers of less importance. All resemble dengue except for shorter duration and usually absence of the recurring phase and the rash. They are caused by viruses, probably transmitted by sandflies, and some may be identical in their etiology and mode of transmission.

58. Diarrheas caused by salmonella.—*a. Etiologic agent.*—May be caused by certain of the organisms comprising the Salmonella (paratyphoid) group.

b. Geographical distribution.—World-wide.

c. Transmission.—Most frequently from human cases or carriers through contamination of food; from the flesh of infected animals, principally cattle and hogs; or from the flesh of healthy animals contaminated during or after slaughtering; also from rats or mice by contamination of exposed food with their excreta; occasionally from raw milk or milk products; and from infected duck eggs. Foods most frequently involved are cold meats, meat salads, twice-cooked hashes, meat sauces, and reheated foods.

d. Recognition.—Incubation period ordinarily 12 to 72 hours but may be shorter. Onset sudden with vomiting, diarrhea, and prostration. Stools may contain blood. Pus seldom found (an important point differentiating bacillary dysentery). Duration 2 to 7 days, occasionally longer.

e. Specific diagnosis.—Isolation of causative organisms from suspected food or from feces of patients early in the disease. While these organisms usually may be readily identified as belonging to the genus *Salmonella*, the identification of individual species often involves complex serological procedures.

f. Treatment.—Symptomatic. As in all cases of acute diarrhea, disturbances in fluid and electrolyte balance occur and these should be corrected by the parenteral administration of fluids when necessay. Purgatives should not be used.

g. Prevention.—Patients should be cared for under enteric precautions if possible. Preventive measures include thorough cooking of meats, proper storage and refrigeration of food stuffs, prohibition of use of reheated hashes and similar foods, and protection of foods from rats, mice, and insects. It is very important to maintain scrupulous cleanliness of kitchens, cooking utensils, and dishes. Food handlers must be instructed in personal hygiene, especially the proper cleansing of hands. Known carriers and persons with diarrhea should be excluded from handling food. (See AR 40-205 and 40-210.)

59. Diarrhea caused by staphylococcus enterotoxin.—*a. Etiologic agent.*—Thought to be a preformed, heat-stable toxin in certain foods and resulting from the growth of certain hemolytic strains of staphylococcus. (Not conclusively proved.)

b. Geographical distribution.—World-wide.

c. Transmission.—Chiefly by foods containing custard or cream fillings, such as eclairs, custards, cream pies, layer cakes, and cream puffs. Raw milk, and hams injected with curing fluid ("tenderized ham") have also caused outbreaks of food poisoning.

d. Recognition.—Onset from 2 to 6 hours after consumption of contaminated food. Onset is sudden, with watery diarrhea, vomiting, abdominal cramps, and prostration. The stools may become bloody. Duration 1 to 3 days. Mortality practically *nil.*

e. Specific diagnosis.—A hemolytic staphylococcus can usually be recovered from the incriminated food. This finding should not preclude the earnest search for Gram-negative pathogens of the *Salmonella* group.

f. Treatment.—Symptomatic, with complete rest in bed, fluid diet until subsidence of symptoms, parenteral fluids in dehydrated patients. Avoid use of purgatives.

g. Prevention.—Absolute cleanliness of kitchens and all cooking utensils; thorough cooking of custards and other fillings; and proper refrigeration of commonly involved foods before and after cooking, are important. Avoid use of filled pastries in the Tropics and during warm seasons elsewhere. Use only pasteurized milk. Food handlers having furuncles or infected lesions on the hands, arms, or face should be removed from duty until their lesions are healed. Isolation of patients is unnecessary, since case-to-case transmission does not occur. (See AR 40–205 and 40–210.)

60. Dysentery, amebic, and amebiasis.—*a. Etiologic agent.—Endamoeba histolytica.*

b. Geographical distribution.—The distribution of this disease probably is world-wide, but infections are much more common in the Tropics and sub-Tropics than in temperate regions. Even in the latter regions the incidence may be high in localities where sanitary conditions are poor.

c. Transmissiion.—Through ingestion of food or drink contaminated with feces containing cysts (and possibly trophozoites) of the causative organism.

d. Recognition.—The clinical picture varies. Some common types are:

(1) *Acute amebic dysentery.*—Onset with abdominal discomfort and diarrhea, gradually increasing in severity until there may be 20 to 40 evacuations daily, with blood and mucus in the stools. Fever and toxemia generally mild.

(2) *Chronic amebic dysentery.*—Usually history of acute attack followed by relapses, with symptoms similar to those of acute attacks but milder.

(3) *Amebic colitis without dysentery.*—Symptoms diverse in character and severity. Diarrhea may alternate with constipation. Symptoms may be those of "irritable colon" and may simulate appendicitis or other surgical conditions.

(4) *Amebic abscess of liver.*—Usually but not always history of dysentery. Onset and course of abscess insidious; pain in liver and right scapula; acute tenderness on percussion over abscess; septic type of fever; leukocytosis.

e. Specific diagnosis.—Depends on demonstrating cysts or motile forms of *E. histolytica* in feces, or the motile forms in abscess contents. In the absence of laboratory facilities a presumptive diagnosis can be made on gross appearance of dysentery stool (blood and glary mucous, not well mixed, and absence of pus) and gross appearance of liver abscess contents (chocolate colored fluid). A therapeutic test, useful when no laboratory facilities are available, is the striking and sometimes complete symptomatic relief occurring in 24 to 48 hours when a patient with acute amebic dysentery is given emetine hydrochloride 0.06 gram (1 grain) subcutaneously on 2 successive days.

f. Treatment.—(1) *For acute or chronic amebic dysentery.*—(*a*) *General care.*—For acute cases, keep patient in bed on liquid diet, broth at first, later add milk, eggs, and custards until acute symptoms subside, then soft, low-residue diet. Supplement diet with four multivitamin capsules daily. Resume full general diet gradually during convalescence.

(*b*) Emetine hydrochloride intramuscularly, 0.03 gram (½ grain) twice a day or 0.06 gram (1 grain) once a day for 4 to 6 days. Toxic effects: lowering of blood pressure, vomiting (controlled by sedative), acute myocardial degeneration, peripheral neuritis (generally from more prolonged treatment than above). Stop

emetine as soon as the dysenteric symtoms have subsided. Concurrently with the administration of emetine give—

(*c*) Carbarsone, 0.25 gram (4 grains) by mouth three times a day for 7 days. Toxic symptoms (rare)—abdominal distress, nausea, vomiting, exfoliative dermatitis (very rare). Follow carbarsone by—

(*d*) Vioform, 0.25 gram (4 grains) by mouth three times a day for 7 days. Toxic symptoms—none. Or (instead of vioform) give—

(*e*) Diodoquin, 0.6 gram (9 grains) by mouth three times a day for 7 days. Toxic symptoms—none. If neither vioform nor diodoquin is available, give chiniofon, 1 gram (15 grains) by mouth three times a day for 7 days. Toxic symptoms—watery diarrhea may occur in ambulatory patients.

(*f*) If treatment is ineffective because of lesions in the lower colon or rectum, give carbarsone or chiniofon by rectum as follows: Carbarsone 2.0 grams (30 grains) dissolved in 200 cc of 2 percent sodium bicarbonate solution. (Carbarsone is insoluble in water.) Give as retention enema at night, following a cleansing enema of 2 percent sodium bicarbonate solution. Retention may be aided by a mild sedative. Give on five consecutive nights, but if it proves irritating, reduce to alternate nights. Or chiniofon (instead of carbarsone) by rectum, 4.0 grams (60 grains) in 200 cc of sterile water, after a cleansing enema of water. Vioform and diodoquin are too insoluble and too irritating to administer by rectum.

(2) *For amebic hepatitis without liver abscess.*—Emetine hydrochloride as above, but continue for 8 days. If evidence of hepatitis persists after 8 days, suspect liver abscess, and if indicated, aspirate (see (3) below). If practicable, check effect of emetine on heart muscle by electrocardiograph before treatment, and daily from fifth day (changes in Q–R–S complex and inversion of T wave). Use chiniofon as above, by mouth, for intestinal infection which probably exists. (Carbarsone may be toxic in some cases of hepatitis.)

(3) *For amebic liver abscess.*—Emetine hydrochloride as above. After 2 to 4 days of emetine treatment the abscess may be drained by aspiration, repeatedly if necessary. Open drainage should be avoided. After completion of the emetine treatment, use chiniofon or vioform as above for the intestinal infection which probably exists.

(4) *For amebic carriers without symptoms.*—(*a*) Carbarsone 0.25 gram (4 grains) by mouth three times a day for 7 days. Toxic symptoms (rare)—see (1) (*c*) above.

(*b*) After completion of carbarsone treatment, give vioform 0.25 gram (4 grains) by mouth three times a day for 7 days. Toxic symptoms—none. Or diodoquin 0.6 gram (9 grains) by mouth three times a day for 7 days. Toxic symptoms—none. If neither vioform nor diodoquin is available, give chiniofon 1 gram (15 grains) by mouth three times a day for 7 days. Toxic symptoms—watery diarrhea may occur in ambulatory patients.

(*c*) Diet soft, if possible. Avoid excessive activity during treatment. Give two to four multivitamin capsules daily as supplement.

g. Prevention.—Drinking water can be made safe by approved methods of sand filtration, but not by the usual method of chlorination alone (superchlorination is required for the destruction of cysts). If an adequately treated water supply is not available, drinking water should be boiled. Troops should be particularly cautioned not to drink raw water from untreated sources. Raw fruits and vegetables should be avoided in areas where human excreta are used for fertilizer, or where they are washed in contaminated ditch water. Known carriers or persons

with any intestinal disturbance should be excluded from handling food. Flies and cockroaches should be excluded from access to food or drink. (See AR 40-205 and 40-210.)

61. Dysentery, bacillary.—*a. Etiologic agent.*—The dysentery bacilli (genus *Shigella*).

b. Geographical distribution.—Bacillary dysentery exists throughout the world; especially common in unsanitary localities. Its incidence is highest in tropical and subtropical regions. Its control is a matter of special importance to armies in the field.

c. Transmission.—Through the ingestion of food or drink contaminated with the feces of cases or carriers of dysentery bacilli.

d. Recognition.—Onset sudden, with fever and abdominal cramps soon followed by passage of loose, yellow or green, watery stools which change to characteristic mucopurulent bloody stools. Tenesmus is a prominent symptom. Toxemia and dehydration are marked in severe cases.

e. Specific diagnosis.—Depends on identification of the etiologic agent in stool cultures.

f. Treatment.—(1) *Chemotherapy.*—(*a*) *Acute bacillary dysentery.*—Sulfaguanadine is the drug of choice. The initial dose is 3.5 grams (52½ grains) followed by 3.5 grams (52½ grains) every 4 hours, day and night, *until the number of stools per day is reduced to five or less.* Then shift to a maintenance dose of 3.5 grams (52½ grains) every 8 hours, day and night, and continue until the stools have been normal for 96 hours. If improvement is not noted in 7 days, discontinue the drug.

(*b*) *Chronic bacillary dysentery.*—Administer 3.5 grams (52½ grains) of sulfaguanadine every 8 hours, day and night. The duration of chemotherapy should not exceed 2 weeks.

(*c*) If sulfaguanadine is not available, use sulfathiazole or sulfadiazine, initial dose 4 grams (60 grains), then 1 gram (15 grains) every 4 hours. If improvement is not noted in 7 days, discontinue the drug. These drugs are more toxic than sulfaguanadine. Sufficient fluids must be administered to insure a daily urinary output of at least 1,500 cc.

(2) *Supportive treatment.*—(*a*) Complete rest in bed.

(*b*) For mild and moderately severe cases without dehydration or severe toxemia, the fluid intake should be 3,000 cc or more every 24 hours.

(*c*) For acute fulminant cases, give intravenous glucose, 50 grams in physiological salt solution 1,000 cc, sufficient to maintain a daily urinary output of 1,000 cc or more. Glucose solutions for intravenous injection should be supplemented with 2 milligrams of thiaminehydrochloride for each 50 grams of glucose.

(*d*) Diet should be fluid, or free from residue.

(*e*) Purgation is not recommended. High enemas and colonic irrigations are also not recommended.

(3) *Serum therapy.*—Monovalent Shiga antitoxin may be of some value in fulminating and markedly toxic cases of Shiga dysentery but it should never preclude the use of sulfaguanadine as described above. Patients should be tested for sensitivity before administering serum. The dose of antitoxin is from 40 to 80 cc given intravenously, twice daily, until temperature falls to normal. The antitoxin should be diluted in 500 cc of normal saline and given very slowly.

g. Prevention.—(1) *General sanitary measures.*—Bacillary dysentery is prevented by the same general sanitary measures employed for preventing other

enteric infections. These include meticulous control of milk, water, and food supplies, proper treatment and disposal of sewage and garbage, and protection of food from flies, and from infectious food handlers. The usual precautions should be taken as regards isolation of patients and sterilization of their feces, clothes, dishes, and other fomites (See AR 40-205 and 40-210).

(2) *Vaccination.*—This has been widely practiced but it is not recommended, as its value has never been satisfactorily demonstrated.

62. Filariasis—bancrofti.—*a. Etiologic agent.—Wuchereria bancrofti.* There is also a sheathed microfilaria of nocturnal periodicity known as *Microfilaria malaya* which is said to be a distinct species.

b. Geographical distribution.—This infection is indigenous in practically all tropical regions of the world. In the Western Hemisphere it is endemic in Central America, South America (Colombia, Venezuela, French, Dutch, and British Guiana, and northern Brazil), and the West Indies. In the United States it has been endemic near Charleston, South Carolina.

c. Transmission.—The adult worms live in the lymphatic system of infected humans, releasing larvae (microfilariae) into the lymph and blood streams. A wide variety of night-biting mosquitoes, including some Anopheles, serve as intermediate hosts. The more important ones are *Culex quinquefasciatus* in many parts of the world, *Culex pipiens* in China, *Mansonioides* species in India, and *Aedes variegatus* in the Pacific Islands. In mosquitoes, the microfilariae, during a period of about 10 days, develop into infective larvae which migrate to the mouth parts, escape when the mosquito bites, penetrate the skin, and start a new infection.

d. Recognition.—In many cases there are no apparent clinical manifestations. When symptoms develop they are due not directly to the filariae but to the blockage of the lymphatic vessels by inflammation and by fibrotic changes around the parasites. After an indefinite, prolonged incubation period, symptoms may be produced by the invasion of lymphatic vessels, most often those of the spermatic cord and groin. The initial acute attack is characterized by lymphangitis, accompanied by usual local signs and fever, and may persist for several days. These attacks recur, and chronic elephantoid manifestations due to blockage of lymph channels tend to develop in the lower extremities and more frequently the scrotum and groin. Hydrocele, lymphatic varicocele, epididymitis, and chyluria also occur. Chyluria may be a prominent symptom after involvement of lymph channels by the parasite. The urine is cloudy, due to the presence of lipoid substances which are soluble in ether. On standing, a clot forms in the urine and a surface pellicle is usually present. The urine also often contains blood, due to rupture of a small vessel into the lymphatic varix which is responsible for chyluria. In these circumstances the night specimens of urine may contain microfilariae.

e. Specific diagnosis.—Depends on finding the microfilariae in blood films. Thick smears may be taken between 9 PM and midnight and stained with Mayer's haemalum or with Giemsa's or Wright's stain. Fresh coverslip preparations sometimes reveal the motile microfilariae.

f. Treatment.—There is no specific treatment. For acute lymphangitis usually caused by hemolytic streptococci, *sulfadiazine* by mouth is recommended. The initial dose is 4 grams (60 grains); subsequent doses 1 gram (15 grains) every 6 hours day and night until the infection is under control. Surgical measures may be indicated for elephantiasis.

g. Prevention.—The prevention of filariasis depends entirely on avoidance of the bites of infective mosquitoes. This depends upon such individual pre-

ventive measures as the proper use of bed nets, repellents, and protective clothing; and upon adequate screening (18-mesh), spray-killing of adult mosquitoes, and eliminating or treating the breeding places of mosquito vectors.

63. Filariasis—onchocerciasis.—*a. Etiologic agent.—Onchocerca volvulus.*

b. Geographical distribution.—Africa, especially in Congo River basin; western slope of Guatemala at altitudes from 2,000 to 6,000 feet; southern Mexico, in States of Chiapas, Oaxaca, Guerrero, and Yucatan.

c. Transmission.—The adult filariae (*O. volvulus*) live in subcutaneous nodules, releasing microfilariae which migrate into adjacent lymphatics and other tissues. The embryos are rarely found in smears from peripheral blood. Various species of black gnats (*Simulium damnosum, S. metallicum,* and others) serve as intermediate hosts, after ingesting microfilaria while feeding on an infected person. In the gnat the microfilariae develop in 6 days into infective larvae which migrate to the mouth parts of the insect, from which they escape while the gnat is feeding. Man is infected by introduction of the parasite into the wound made by the insect. The parasite then develops to the adult stage in from 2 months to a year and is found in the subcutaneous tumors.

d. Recognition.—(1) Formation of tender, freely movable and easily enucleated subcutaneous nodules (diameter 6 to 30 millimeter) in scalp, trunk, axilla, popliteal space, and vicinity of the elbow.

(2) In about 5 percent of cases, ocular complications follow migration of microfilariae into the eye, producing keratitis, iritis, and conjunctivitis. Photophobia, zerosis, impairment of vision, sometimes complete blindness, may follow.

(3) Generalized lichenoid dermatitis may occur, with intense itching, particularly at night.

e. Specific diagnosis.—(1) Identification of microfilariae in fluid aspirated from center of nodules, or in tissue of an excised nodule, or in a biopsy specimen of adjacent tissues.

(2) Demonstration of the threadlike adult worm in excised nodules.

f. Treatment.—Enucleation of nodules.

g. Prevention.—The transmitting gnats (*Simulium*) breed in streams, the larvae and pupae being attached to rocks or plants beneath the surface. No method of control of breeding has been devised. The adults bite only during daylight and may be deterred by head nets, protective clothing, and chemical repellents applied to the skin. Smoke smudges and pyrethrum sprays may repel the gnats from the vicinity of personnel in encampments and buildings.

64. Filariasis—loaiasis.—*a. Etiologic agent.—Loa loa.*

b. Geographical distribution.—Tropical West Africa, especially Congo River basin.

c. Transmission.—Adult worms living in subcutaneous tissue liberate microfilariae which enter blood stream. Transmitted to man by blood-sucking mango flies (*Chrysops*), which bite only in daylight, preferably in shade.

d. Recognition.—Usually no symptoms except when adult worms migrate under the ocular conjunctiva or in the skin. This may be accompanied by local pain, swelling, and inflammation. Subcutaneous migratory swellings (Calabar swellings) up to size of hen's egg, may appear in the skin. Their relation to the infection is not completely understood.

e. Specific diagnosis.—Demonstration of the microfilariae in the blood as for *W. bancrofti*, except that periodicity of *Loa loa* is diurnal. Sometimes the adult worm may be identified after extraction.

f. Treatment.—Surgical removal of the worm. There is no specific drug.

g. Prevention.—Protection from bite of *Chrysops*, by protective clothing, nets, and repellents.

65. Hookworm infection.—*a. Etiologic agent.*—*Necator americanus* is almost exclusively the hookworm of man in the Western Hemisphere. *Ancylostoma duodenale* and *Necator* are both prevalent in the Eastern Hemisphere. The dog hookworms *Ancylostoma braziliense* and *A. caninum* infect the skin of man in their larval stage, causing the disease known as "creeping eruption." (This infection is acquired usually by lying on a sandy place, such as a beach, which has been contaminated by dog excreta.)

b. Geographical distribution.—In the United States hookworm infection occurs in the South from Virginia and Kentucky to eastern Texas. The rural population of some counties in southern South Carolina, Georgia, Alabama, and Mississippi, and in northern Florida, still show an incidence of 50 percent or more. Hookworm infection is prevalent in the Tropics wherever the climate is moist and sanitary disposal of feces is not practiced.

c. Transmission.—The hookworms affecting man live in the small intestine and suck blood from the mucosa. Eggs are passed in the feces and hatch in soil. Infective larvae develop in a few days, penetrate the skin of man, and reach the intestine via lungs, trachea, and esophagus.

d. Recognition.—In well nourished adults it requires 50 or more worms to produce clinical symptoms such as lassitude, fatigue, weakness, and pallor. "Ground itch" is often an early symptom. There may be secondary anemia, slight or moderate eosinophilia, and edema.

e. Specific diagnosis.—Identification of the characteristic ova in the feces. The ova of the two common species cannot be differentiated morphologically.

f. Treatment.—(1) *General.*—Give a light meal, preferably free from fats, the night before instituting treatment. No preliminary purge is necessary unless constipation exists. Give drug in the morning on an empty stomach. Avoid food for 2 hours after treatment, and alcohol for 24 hours before and after treatment. A saline purge should be given the following day if bowels have not moved since treatment.

(2) *Drugs.*—(*a*) In the *absence* of *Ascaris* infection, use tetrachlorethylene: adult dose 3 cc in hard gelatin capsules, followed in 2 hours by a saline purge. Drug sometimes causes dizziness and drowsiness, but no case of fatal toxicity has been reported.

(*b*) In the *presence* of *Ascaris* infection, use hexylresorcinol followed by tetrachlorethylene.

Hexylresorcinol crystoids (caprocol) : adult dose 1 gram (15 grains) on empty stomach in morning. Avoid all food for 4 hours after treatment (*very important*). Toxic symptoms—none, except burning of mouth if crystoid is chewed. This treatment will usually remove all ascaris worms and about 50 percent of hookworms. It should be followed after 3 days by treatment with tetrachlorethylene to remove the remainder of the hookworms.

(*c*) Iron should be administered to all hookworm cases showing anemia. An effective preparation is ferrous sulphate (desiccated) in capsules; adult dose 0.35 gram (5 grains) three times a day after meals.

(3) *Diet.*—Should be rich in iron and vitamins.

(4) *Post-treatment stool examination.*—Should be made 1 week after treatment. If eggs are found, treatment should be repeated until cure is obtained.

(5) *Treatment of "creeping eruption."*—Use one of the following methods. Repeated applications may be necessary to obtain cure.

(*a*) Saturate cotton with ethyl acetate; apply to area just beyond advancing edge of skin lesion, and cover with adhesive tape for 24 hours.

(*b*) Freeze lightly with ethyl chloride spray or dry ice on area 1 inch in width just beyond advancing edge of skin lesion.

g. Prevention.—Provide for sanitary disposal of excreta and protect the skin, especially the feet, against contact with soil contaminated with hookworm larvae.

66. Leishmaniasis—kala-azar (visceral leishmaniasis).—*a. Etiologic agent.*—*Leishmania donovani.*

b. Geographical distribution.—The disease is widespread, and occurs in Mediterranean countries, south Russia, India, China, Manchuria, Abyssinia, Sudan, northern and eastern Brazil, Chaco Region of Argentina, Paraguay-Brazil border (Matto Grosso), and northern Bolivia (Yungas).

c. Transmission.—The available evidence suggests that the disease is transmitted by the bites of infected sandflies of the genus *Phlebotomus* and possibly through the ingestion of food or drink contaminated with *L. donovani*, or by contact. Dogs and other lower animals are susceptible to infection.

d. Recognition.—The principal symptoms are:

(1) An irregular, recurring fever over a period of weeks, sometimes having a double daily rise.

(2) Progressive enlargement of spleen, later of the liver.

(3) Anemia, leukopenia, and emaciation.

(4) Dysentery or diarrhea.

e. Specific diagnosis.—By demonstration of *L. donovani* either in smears or cultures of the peripheral blood or by material obtained by spleen, liver, or bone marrow puncture.

f. Treatment—(1) *Pentavalent antimony compounds.*—A number of effective pentavalent antimony compounds have been developed, including neostam (stibamine glucoside), solustibosan (Bayer 561), and neostibosan (Bayer 693). The last named is least toxic and most effective, and its use is recommended. It is given intravenously, 15 doses on alternate days, the first dose 0.2 gram and subsequent doses 0.3 gram. Use this drug as a 10 percent solution freshly prepared from sterile distilled water. The dosage and method of administration of *Neostam* is the same as for *Neostibosan.* (The trivalent compounds of antimony, such as fouadin (neoantimosan), are relatively ineffective in visceral leishmaniasis.)

(2) *Potassium antimony tartrate* (USP).—A 2 percent freshly prepared solution intravenously on alternate days. Initial dose 2.0 cc (0.04 gram), and each succeeding dose increased by 1.0 cc until 5.0 cc are being given. Continue until a total of 40 or more doses have been given. If toxic symptoms develop, reduce dose. Toxic symptoms of antimony drugs: coughing immediately after administration (not important); nausea, vomiting, dizziness, collapse.

(3) In the Egyptian Sudan and some adjacent areas leishmaniasis is very resistant to treatment with antimony preparations. British investigators have recently reported that antimony resistant cases show a good response to intravenous injections of stilbamidine isethionate (4:4 diamidine stilbene isethionate). The drug must be used in a *freshly prepared solution* in 10 cc of sterile distilled water

without heating. The water must be neutral or very slightly acid (pH 6.8–7.0). Old solutions may cause severe late toxic effects on the liver, kidneys, or pancreas, even after completion of a course of treatment. The dose is 1.0 milligram (0.001 gram) per kilogram of body weight (maximum adult dose 0.15 gram) every other day for 15 injections. Repeat this course of treatment *only* if cure is not obtained and *after interval of 1 month.* Some cases show fall of blood pressure and syncope after first or second injection. This may be prevented or relieved by injection of a small dose of epinephrin. The administration of calcium and glucose during treatment may protect the liver from damage.

NOTE.—Stilbamadine isethionate is not available for general distribution.

(4) *Diet.*—The diet may contain whatever foods the patient can tolerate. It probably is wise to supplement it with vitamin B complex and, if it contains less than 6 ounces of citrus fruit juice, ascorbic acid should be given in doses of 50 milligrams per day.

g. Prevention.—In endemic areas antisandfly measures should be used, such as elimination of breeding places and the use of fine mesh bed nets, insect repellents, and insecticide sprays. Avoid unnecessary contact with native dogs. Segregate and treat effectively all cases of the malady.

67. Leishmaniasis—oriental sore (Old World cutaneous leishmaniasis).—*a. Etiologic agent.—Leishmania tropica.*

b. Geographical distribution.—Apparently this disease does not occur commonly in localities where kala-azar is present. It occurs in Mediterranean countries, central Asia, India, China (Hunan), southern Russia, and many parts of Africa.

c. Transmission.—As in the case of kala-azar, sandflies of the genus *Phlebotomus* are suspected as vectors. As the disease can be transferred by inoculation, the possibility of contact infect must also be considered.

d. Recognition.—The disease begins as a small itching papule on exposed sites. The lesion gradually extends and ulcerates. Lesions may be multiple, but there is no general dissemination.

e. Specific diagnosis.—The diagnosis depends on the demonstration of *L. tropica* in stained films or cultures of material aspirated from the indurated zone surrounding the ulcer.

f. Treatment.—Local treatment may be tried first, consisting of topical applications of dry ice or injection of 2 cc of 1 percent solution of berberine sulfate into the edges of the sores. If these measures are not effective, intravenous injections of neostibosan may be tried, using the same dosage as for visceral leishmaniasis.

g. Prevention.—Take the necessary precautions to avoid the transfer of infectious materials by insects or contact.

68. Leishmaniasis—espundia (American mucocutaneous leishmaniasis).—*a. Etiologic agent.—Leishmania brasiliensis.*

b. Geographical distribution.—Mexico, Central America, South America (Argentina, Bolivia, Brazil, Colombia, Ecuador, the Guianas, Paraguay, Peru, and Venezuela). The disease is said to be common in forest regions among collectors of chicle gum and rubber. Has been encountered in American soldiers in Panama, causing chronic ulcerations of the skin.

c. Transmission.—The exact method is unknown, but it is suspected that sandflies of the genus *Phlebotomus* may act as vectors, and also that contact infections may occur.

d. Recognition.—Commences as small papule, on arms or often on margins of ears; extends and may suppurate. Later, ulcers appear around margins of nose and mouth; these may extend and cause extensive destruction of the tissue in naso-oral region. There is no general dissemination. If untreated, death may occur from sepsis and exhaustion.

e. Specific diagnosis.—The diagnosis depends on demonstration of *L. brasiliensis* in cultures or smears of material obtained by puncture of the edge of the initial ulcer or in material from nodules or ulcerations in the mucous membranes.

f. Treatment.—The treatment of the initial lesion is the same as that outlined for oriental sore above. During the stage of mucous membrane involvement, use the treatment recommended for kala-azar.

g. Prevention.—Use methods similar to those advocated above for the control of kala-azar and oriental sore.

69. Malaria.—*a. Etiologic agents.*—*Plasmodium vivax*, causing benign tertian malaria; *P. falciparum*, causing malignant tertian (aestivo-autumnal) malaria; *P. malariae*, causing quartan malaria; and *P. ovale*, causing a mild type of tertian malaria.

b. Geographic distribution.—The distribution of malaria is almost world-wide but there is considerable variation in the geographic distribution of the different species of malaria parasites. *P. vivax*, which is most widely distributed, occurs between 45° north and 45° south latitude and is the most common species found in the Temperate Zones. *P. falciparum* is confined usually to the Tropics and sub-Tropics, and in many tropical regions it is the most common species. It rarely occurs where the average summer temperature is lower than 70° F. or the winter temperature cooler than 48° F. *P. malariae* and *P. ovale* are relatively uncommon and their distribution is irregular.

c. Transmission.—From man to man through the agency of various species of anopheline mosquitoes, the relative importance of which varies in different regions.

d. Recognition.—To be suspected not only in patients with periodic chills and fevers but also in any obscure illness, febrile or nonfebrile, in endemic regions. The symptoms of malaria may vary in different cases from mild headache or diarrhea to severe chills and fever, and to delirium or coma. Exposure, surgical operations, and shock may precipitate a relapse in latent infections.

e. Specific diagnosis.—(1) By demonstration of the plasmodia in blood. In each suspected case, examine the blood as soon as possible, preferably by thick smear. Thin smears should also be taken for use when species diagnosis cannot be made in thick smears. If malaria parasites are not found, take smears on successive days, because the symptoms in persons suffering from a first attack may be initiated by a low density of parasites, and because in *falciparum* infections there may be very few parasites in the circulating blood during the second 24 hours of each asexual cycle. Suspect as malaria infection all febrile illnesses occurring in endemic areas; suspect a recurrence or relapse in all who give a history of previous malaria.

(2) To obtain thick smears the surface at the end third of a clean and grease-free slide is placed in contact with the crest of a drop of welling blood and gently rotated thereon without touching the skin, until the blood is smeared over a circular area about the size of a dime. Allow the smear to air-dry in a horizontal position protected from insects and dust. Dry thick films may be stained by the Giemsa method or with Field's stain, if available. They should not be called negative until carefully examined for 5 minutes.

(3) To obtain thin smears stain with Wright's or Giemsa's method. Should not be called negative until carefully examined for 15 minutes.

(4) In *P. falciparum* infections estimate the proportion of infected erythrocytes if possible. If 5 percent or more erythrocytes are infected, treat as you would a comatose patient (see below). In *P. falciparum* infections the demonstration of parasites in the blood is difficult at times, even in the presence of a severe infection with cerebral symptoms, including coma. When in endemic areas, suspect as *P. falciparum* malaria every case of febrile illness in which coma or medical shock occurs. Headache, fever, and prostration are frequently the only prodromal symptoms of cerebral malaria. This form may simulate acute alcoholism; or the patient may be maniacal, requiring morphine. During the stage of onset the temperature is often little elevated and in the *presence of coma it may be normal or subnormal.* If facilities for immediate examination of blood smears are not available for confirmation of the diagnosis in such emergency cases, immediate malaria therapy should be initiated.

(5) Trauma and surgical procedures frequently precipitate serious recurrence of malaria. Under these circumstances in endemic regions, malaria should be suspected if fever occurs. All such cases occurring under circumstances in which blood examination cannot be made should receive immediate malaria treatment.

f. Treatment.—(1) *Medication.*—(*a*) *Malaria—uncomplicated* (patient able to retain oral medication).

1. Combined QAP treatment (method of choice).—(a) Quinine sulfate, 0.64 gram (10 grains) three times daily after meals for 2 or 3 days, or until pyrexia is controlled. Then give

(b) Atabrine, 0.1 gram (1½ grains) three times daily after meals for 5 days. Then after 2 days without antimalarial medication, give

(c) Plasmochin, 0.01 gram (3/20 grain) three times daily after meals for 5 days, except for the debilitated patient, who should receive only two doses daily. (Discontinue if toxic symptoms occur. Never give atabrine and plasmochin concurrently.)

2. Atabrine-plasmochin treatment.—Atabrine, as above, for 7 days. Then after 2 days without antimalarial medication, give *plasmochin*, 0.01 gram three times daily for 5 days, as above. (May be used for simple *vivax* infections and in other infections when no quinine is available.)

3. Quinine-plasmochin treatment.—Quinine sulfate, as above, for 7 days, during the last 5 of which accompany each dose of quinine with plasmochine, 0.01 gram three times daily. (Method when no atabrine is available.)

NOTE.—Quinine sulfate tablets are more quickly effective if dissolved before taken. Ten grains of quinine sulfate may be dissolved in 1 ounce of water which has been acidified by the addition of 10 minims of *dilute sulfuric* or *hydrochloric acid* (USP), or 2 grams (30 grains) citric acid.

(*b*) *Malaria with coma.*—Quinine dihydrochloride, 0.64 gram (10 grains) in at least 200 cc sterile normal saline given intravenously *very slowly* every 8 hours until patient can retain oral medication; then treat as an uncomplicated case. (See (*a*) above.)

(*c*) *Malaria with vomiting* (unable to retain oral medication).

1. Quinine dihydrochloride, 1.0 gram (15 grains) in 10 cc sterile normal saline injected carefully into the gluteal muscles of one buttock; site

of injection then massaged thoroughly for 2 minutes. Care must be taken to avoid the sciatic nerve. Dose may be repeated if necessary in 8 hours in other buttock. As soon as patient can retain oral medication, treat as an uncomplicated case. (See (*a*) above.)

2. *Atabrine dihydrochloride*, 0.2 gram (3 grains) in 5 cc sterile normal saline may be substituted for intramuscular injection if no quinine dihydrochloride is available.

(2) *Control of vomiting.*—Do not allow solid food just before a febrile paroxysm is expected. If there is nausea or vomiting, sips of alkaline water may be helpful. If vomiting occurs soon after a dose of quinine, the dose should be repeated at once. There is a good chance that it will be retained without further difficulty.

(*a*) If the vomiting is frequent and troublesome, the intravenous administration of *glucose* is of particular value inasmuch as many patients who are vomiting develop varying degrees of acidosis, as evidenced by the presence of acetone bodies in the urine. Glucose may be given by the drip method (preferable), 500 cc of 5 percent glucose in isotonic saline being infused over a period of time in the usual way, or 50 cc of 50 percent glucose in saline may be injected slowly from the syringe. In either case the glucose may be repeated several hours later if it seems desirable. Glucose solution for intravenous injection should be supplemented with 2 milligrams of thiamine hydrochloride for each 50 grams of glucose.

(*b*) Even though vomiting has not occurred, certain malaria patients receiving oral therapy may fail to absorb the drugs. For this reason, patients who fail to respond within 36 hours to orally administered specific drugs should receive intravenous or intramuscular therapy (see above) until such time as a therapeutic response is obtained. This is usually evidenced by rapid improvement, patient becoming rational and regaining the ability to ingest and absorb food and drugs.

(3) *General care.*—Keep patient in bed. Discomfort from high temperature may be relieved by cold sponges and wet packs and the administration of acetylsalicylic acid. An elevation of temperature above 104° F. in the paroxysms of *vivax* and *quartan* malaria is uncomfortable but not dangerous. On the other hand, this degree of fever is cause for concern in *falciparum* infections and is an indication for a temporary increase in the dose of the antimalarial drug employed. Iron and arsenic have long been regarded as having value in the treatment of anemia which may result from an attack of malaria.

(4) *General discussion of therapy.*—While treatment should be initiated as soon as a diagnosis is effected, it is desirable to time one of the daily doses of quinine so that it is administered about 1 hour before the period when a paroxysm is anticipated.

(*a*) In general, one may expect that the doses recommended will cause all trophozoites of *P. vivax* and *P. malariae* to disappear from the peripheral blood after the third day of administration. In the case of *P. falciparum* larger doses may be required to produce analogous results. Considerable difference has been noted in the response of different strains of the latter parasite to antimalarial drugs. If larger amounts of quinine are required, a fourth daily dose may be administered.

(*b*) Quinine and atabrine exercise a parasiticidal action on the trophozoites of all species of the human malaria parasites, and on the gametocytes of some,

but are not lethal to the gametocytes of *P. falciparum.* Plasmochin exerts some slight action on the trophozoites of *P. malariae* and *P. vivax*, but its most striking action is upon the gametocytes of *P. falciparum*, which it devitalizes and renders noninfectious. When the latter drug is administered, as described above, the relapse rate appears to be substantially lowered. The simultaneous or *concurrent administration of atabrine and plasmochin* appears to aggravate the toxicity of each and *must be avoided.*

(*c*) In patients experiencing definite quotidian (daily) paroxysms, one should not expect the early doses of these drugs to prevent the occurrence of the next anticipated paroxysm, nor even the paroxysm expected the following day. However, if the drugs are adequately absorbed, suppression of the paroxysms on the third and subsequent days may be expected. If the paroxysms of *falciparum* malaria tend to be prolonged and the fever ascends above 104° F., additional doses of quinine (0.6 grams; 10 grains) may be administered every 3 hours until the temperature descends.

g. Prevention (see AR 40–205 and 40–210).—Malaria preventive measures should be based so far as possible on the specific epidemiology of malaria in the area to be controlled. Military necessity may sharply limit the preliminary malaria survey but as much information as possible should be obtained about the amount and type of malaria and about the specific local vectors and their habits. Control measures may be directed against the mosquito or the plasmodium and in any situation all practical devices should be employed. It is important to have specific plans and spot maps wherever possible.

(1) *In fixed installations.*—There are seven general measures available to protect troops in fixed installations, as follows:

(*a*) The correct siting of camps. When feasible, camps should be on elevated ground and at least a mile from important breeding places of the local vector and from native villages.

(*b*) The spray-killing and repelling of adult mosquitoes by using approved pyrethrum insecticidal sprays not only in the military buildings but, if possible, in surrounding civilian dwellings. This measure is often immedately effectve in controlling malaria.

(*c*) The intensive use of proper screening (16- or 18-mesh, with aperatures not larger than 0.0475 inch diameter) and bed nets (a *stiff* bobbinet having holes not over 0.0500 inch diameter). To keep out sandflies the apertures of the net should not exceed 0.0334 inch diameter.

(*d*) The routine application of such antilarval measures as oiling, Paris greening, draining, filling, emptying, channeling, and shading. It must be noted that antilarval measures are never *immediately* effective in controlling malaria.

(*e*) Systematic education and training of all ranks in order to develop and maintain effective *malaria discipline.*

(*f*) Use of approved repellent lotions (indalone or 612) and protective clothing, such as head nets, gloves, and long trousers and sleeves.

(*g*) Use of quinine and atabrine prophylaxis. This is usually least important at base camps where mosquito control has been established.

(2) *In field operations.*—In field operations malaria control must depend chiefly on prophylactic drugs, repellent lotions, protective clothing, and sleeping nets, none of which is entirely satisfactory. In some field situations the use of pyrethrum insecticide sprays may be very helpful.

h. Clinical prophylaxis (suppressive treatment).—(1) *Atabrine.*—Give 0.1 gram (1½ grains) twice daily after meals on 2 days a week, allowing a 2- or 3-day interval between days of medication. Continue only while need for emergency suppressive treatment exists. (See note below.) Then watch for indications of malaria and, if required, give curative treatment as in *f* above.

(2) *Quinine.*—If no atabrine is available, give quinine sulphate 0.64 gram (10 grains) after the evening meal each day while need for emergency suppressive treatment exists. (See note below.) Then discontinue, observe for indications of malaria, and if required, give curative treatment as in *f* above.

NOTE.—There is no drug which in safe doses will prevent mosquito-borne infection with malaria. However, quinine and atabrine, in small doses, are useful in suppressing the appearance of clinical symptoms after infection. They are almost equally effective. Such suppressive treatment will enable malaria infected troops to maneuver and fight actively in spite of an infection which otherwise would incapacitate them. When these troops stop taking suppressive treatment many of them may develop clinical malaria and require therapeutic treatment. It may be wise to stagger the terminal point of prophylactic medication so that hospital facilities are not overtaxed when a large force returns from a hyperendemic area.

70. Blackwater fever.—*a. Etiologic agent.*—Not definitely known but generally believed to be associated with repeated attacks of *falciparum* malaria in hyperendemic areas. Predisposing factors seem to include chilling, trauma, alcoholism, fatigue, and perhaps the irregular and inadequate use of quinine.

b. Geographical distribution.—Occurs in most areas where malaria is hyperendemic.

c. Recognition.—Onset sudden, with persistent fever. The three cardinal symptoms are hemoglobinuria, fever, and icterus. Temperature may reach 102° F. to 103° F., usually accompanied by chill. Other symptoms are repeated vomiting of bile-stained fluid, abdominal pain, jaundice or subicteric sclerae, enlarged tender liver and spleen, severe prostration, progressive severe anemia, elevated plasma bilirubin, with an increased indirect Van den Bergh reaction which may later become direct. There is usually some degree of urinary suppression. Color of urine may vary from light red to black (hemoglobinuria). Asexual forms of *P. falciparum* usually absent from peripheral blood within 2 to 3 hours of onset. Crescents and forms of tertian and quartan may be present. There may be urinary suppression due to precipitation of acid hematin in renal tubules. This may occur early and suddenly, and is the commonest cause of death, if not corrected promptly. Cardiac failure is a common cause of death in posthemolytic period. Other complications: grave anemia, hyperpyrexia, uncontrollable vomiting, hiccough, sudden drop in temperature, prostration, and coma.

d. Specific diagnosis.—None. *P. falciparum* may be found in blood smears taken either before or after, but rarely during an attack.

e. Treatment.—(1) Do not give quinine or atabrine until convalescence from the attack of blackwater fever is established.

(2) Absolute rest. Keep patient warm. Whenever possible, treat the patient at the place where he was taken ill until definite improvement occurs.

(3) Give a minimum of 2,000 cc of fluids per day and up to 6,000 cc if possible. Volume of urine should be 1,200 to 1,500 cc per 24 hours. If unable to void, catheterize every 4 hours in order to determine urine output and reaction to litmus.

(4) Immediate transfusion of whole blood if the patient is not anuric. This may be repeated daily as indicated by persistence of severe hemoglobinuria or severe anemia and asthenia.

(5) During period of vomiting, if urine is acid or if anuria exists, give 1,000 cc of sodium bicarbonate solution intravenously as described under cholera treatment. Repeat after 12 hours if urine remains acid. When vomiting is controlled, if urine is acid give sodium bicarbonate 0.6 gram (10 grains) by mouth every 1 to 2 hours until urine is alkaline to litmus, and thereafter in sufficient amounts to maintain slightly alkaline reaction.

(6) If plasmodia are found in blood smear *after* convalescence, give atabrine 0.1 gram the first day, two 0.1-gram doses the second day, and three 0.1-gram doses the third day. Continue this dosage for 5 additional days. Watch for recurrence of hemoglobinuria, as atabrine has occasionally precipitated an attack.

f. Prevention.—(1) Adequate treatment of malaria cases, especially when due to *P. falciparum.*

(2) Change of residence to temperature climate if feasible. Blackwater fever often recurs if patient remains in a malarious region.

(3) Recognition of preblackwater state is important. Characteristics: toxic appearance, slight jaundice, enlarged tender liver, dark-colored urine due to excessive urobilin. Supplement antimalarial therapy with alkalis to maintain the urine alkaline.

71. Oroya fever.—*a. Etiologic agent.—Bartonella bacilliformis.* The chronic stage of oroya fever is verruga peruana. The acute stage as a rule and sometimes both stages together are referred to as bartonellosis.

b. Geographic distribution.—Endemic areas are confined to hot, narrow valleys of Peru, Ecuador, and Colombia, and possibly northern Chile and western Bolivia, at altitudes from 1,500 to 9,000 feet.

c. Transmission.—The causative agent probably is transmitted by sandflies of the genus Phlebotomus, biting principally at night from January to April. Native dogs with bartonellosis may be an animal reservoir of infection.

d. Recognition.—(1) *Early, acute stage* (oroya fever).—Incubation period is about 3 weeks. Insidious prodromal period is usually characterized by malaise and low fever. Course of disease is then severe, with high, irregular, remittent fever. Marked anemia develops rapidly, with intense pain over the long bones. Death occurs in 10 to 40 percent of cases during the second or third week of the disease.

(2) *Late, chronic stage* (verruga peruana).—Generally follows attack of oroya fever by 30 to 60 days, or more, but may occur without an oroya fever stage. Initially there is fever. Severe rheumatic pains frequently occur. Miliary (lenticulate or nodular, often granulomatous) eruption develops on the face, trunk, and extensor surface of extremities. Lesions may develop on all mucous surfaces and the lesions may bleed freely or become gangrenous. Mortality is nil.

e. Specific diagnosis.—(1) *Oroya fever.*—(*a*) Identification of organism in stained blood films.

(*b*) Blood culture.

(2) *Verruga peruana.*—(*a*) Usually history of preliminary attack of oroya fever.

(*b*) Characteristic lesions.

f. Treatment.—There is no specific treatment. Multivitamin capsules and iron should be administered during convalescence.

g. Prevention.—In endemic areas avoid unnecessary exposure to bites of sandflies, especially at dusk and dawn. Sleep under fine mesh bed net. Spray tents and quarters, and especially the bed net, with pyrethrum insecticide at dusk. Apply approved mosquito repellent skin lotions, as these are also effective against sandflies. In some endemic areas it may be feasible to plan travel so as to camp at night at an altitude above 9,000 feet or below 1,500 feet (outside of the phlebotomus zone).

71.1. **Pinta (carate).**—*a. Etiologic agent.*—Various fungi have been suspected but since 1938 a spirochete, *Treponema carateum*, morphologically identical with *T. pallidum*, has been accepted by some authorities as the causative agent. It may be found in tissue juice from skin lesions and lymph nodes.

b. Geographical distribution.—Mexico, West Indies, Central America, and South America.

c. Transmission.—The disease occurs in damp, low-lying tropical regions. It affects the dark races chiefly and is rare in whites. It occurs most often in young adults from 15 to 25. There is no proof of congenital transmission, and it is apparently not contagious by person-to-person contact. Transmission may be by the bite of a blood-sucking insect (perhaps by a species of *Simulium* in some cases).

d. Recognition.—The initial lesion appears at the site of the port of entry of infection as a minute papule which appears 7 to 10 days after inoculation. Distribution of skin lesions is localized at outset; later the lesions may be generalized. Eruption is often symmetrical, at times strikingly so. In extremely rare cases it is unilateral. Location of the eruption is usually on face and extremities; often over bony prominences, such as knuckles and malleoli. It may affect any part of skin except the scalp. Palms, soles, and genitals are rarely affected. Small patches may be present on mucous membranes. Color of skin lesions is usually a shade of blue (slaty or leaden). Bluish color may be either diffuse or stippled. When blue pigmentation eventually disappears there is left a partial depigmentation which may go on to complete depigmentation, simulating vitiligo. Course of disease is extremely chronic.

e. Specific diagnosis.—Demonstration of the causative agent in material from typical lesions. According to Leon y Blanco, positive Wassermann reactions were not obtained prior to the development of secondary lesions, and only in the advanced cases with marked pigmentation did the positive tests approach 100 percent.

f. Treatment.—As for yaws. Optimum amount and duration of treatment is not yet established.

g. Prevention.—Mass treatment of infected individuals. Avoidance of contact with infective lesions. Precautions to avoid the bites of flies and gnats.

71.2. **Plague.**—*a. Etiologic agent.*—*Pasteurella pestis.*

b. Geographical distribution.—Endemic in India, Indo-China, the East Indies, New Caledonia, Central China, Manchuria, and Mongolia. Less important foci of endemic infection exist in Africa (Morocco, Egypt, Uganda, Angola, Kenya, Madagascar, and the Belgian Congo), in South America (Brazil, Ecuador, Peru, Bolivia, and Argentina), and in the Azores. Sporadic cases have occurred in almost all the major seaports of the world, including those of the United States.

c. Transmission.—(1) Plague occurs as an epizootic disease among *domestic* rats, and is transmitted from rat to rat and from rat to man by certain fleas (*Xenopsylla cheopis* and others). Infection of man may occur by regurgitation of *P. pestis* into the wound when the flea bites, or by inoculation of the infective

feces of the flea. Plague in humans exists largely because of the domestic habits of rats and its occurrence as an endemic or epidemic disease is largely conditioned by the density of the rat population.

(2) In addition to flea-borne transmission, infection may result from handling infected rodents or infectious tissues.

(3) It must also be remembered that human cases of plague may disseminate the disease directly by droplet infection or by expectoration or by discharge from buboes.

d. Recognition.—Incubation period is usually 3 to 7 days, occasionally longer. Case fatality rate high. There are three chief clinical types: (1) *Bubonic* (most common).—Characterized by enlargement of lymph nodes (buboes) draining the area of the flea bite. There is usually a rapid and severe course, with high fever and great prostration.

(2) *Septicemic* (less common).—May be generalized lymph node enlargement, subcutaneous hemorrhages, and pustules. Course is rapid and overwhelming, generally fatal.

(3) *Pneumonic.*—Clinical picture is that of a virulent septic pnuemonia; fulminating course; thin, mucoid, bloody sputum; almost invariably fatal. Plague pneumonia may develop in bubonic cases, or from handling infected rodents or by droplet infection from another case.

e. Specific diagnosis.—(1) Smear of aspirated bubo contents or sputum stained with methylene blue shows short bacilli (*Pasteurella pestis*) with bipolar staining and sometimes with swollen, vacuolated, involution forms.

(2) Culture from bubo, blood, or sputum on nutrient agar or in broth shows characteristic Gram-negative organisms.

(3) Inoculation of bubo juice, blood, or sputum into mice, rats, or guinea pigs, intraperitoneally, or by skin scarification, produces death in 24 to 72 hours. At autopsy of animal, characteristic lesions are found and *P. pestis* can be recovered.

***Warning:** All infectious material must be handled with the greatest care.* All animals inoculated for plague diagnosis *must* be free of fleas and other ectoparasites prior to inoculation and thereafter kept in insect-proof cages in a separate room from other animals. All persons handling smears, cultures, cages, or inoculated animals must wear gowns, rubber gloves, and masks, and must observe the strictest aseptic technique.

f. Treatment.—(1) *General.*—Morphine as indicated for restlessness and delirium. Force fluids by mouth or parenterally for toxemia.

(2) *Chemotherapy.*—Sulfathiazole by mouth as follows: Initial dose 4.0 grams (60 grains); subsequent doses 1.5 grams (22½ grains) every 4 hours day and night until temperature has been normal for 5 to 7 days. It should be borne in mind that the toxic effects of these large doses may mask clinical improvement in the disease. In fulminating cases intravenous sodium sulfathiazole (5 percent solution in sterile distilled water) may be tried as follows: Initial dose, 0.06 gram (1 grain) per kilo of body weight given slowly. Subsequent doses 0.03 gram per kilo every 6 hours. Change to oral dosage as soon as possible. The use of sulfadiazine in man has not been reported to date (December 1942), but in mice it is more effective and less toxic than sulfathiazole.

(3) *Surgical.*—Hot, wet applications to the bubo may hasten localization of the infection. Incision should be delayed until localization is complete in order to avoid blood stream infection.

g. Prevention.—(1) *Isolation of patient.—This is imperative.* Keep patient in separate screened room and allow only attendants to enter. Burn all waste articles contaminated by discharges. In pneumonic or suspected pneumonic cases, attendants must wear hoods with goggles or plastic eye openings, coveralls or complete gown with trousers, and rubber gloves. Sterilize all bedding, linens, and utensils in contact with patient, by boiling or autoclaving. Wash walls and floor of room and all furniture with 5 percent cresol compound after discharge of case and allow room to air for 48 hours. Handle bodies of persons dying of plague with strict antiseptic precautions.

(2) *Sanitary measures.*—(*a*) Ratproof buildings and ships.

(*b*) Carry out rat extermination program by trapping and poisoning with red squill, or by fumigation with methyl bromide or cyanide gas. Cyanide fumigation should be performed only by thoroughly trained personnel.

(*c*) Keep under constant surveillance the incidence of plague in the rat population of port and other cities of the endemic areas. "Plague in rats usually precedes plague in humans."

(*d*) Constant vigilance should be exercised in and around camps to prevent rodent harborages and access of rats and other wild rodents to food supplies, special attention being given to the thorough collection and proper disposal of garbage.

(*e*) If a bubonic case occurs, determine source of infection. If source is in a city or town, trap and examine rats from focus of infection outward until no more plague-infected rats are found. Then trap and poison from periphery of infected area to center and follow at once with ratproofing and destruction of rat harborages.

(*f*) If a pneumonic case occurs, quarantine all contacts under guard, and take temperatures every 12 hours for 7 days. Also quarantine under military or police guard the area where the infection was acquired and where contacts may have occurred, prohibit entrance and exit of inhabitants, and make house-to-house inspection, taking temperatures twice daily until 7 days after last case is discovered, isolating all persons developing fever regardless of cause. Guards should be cautioned to avoid close contact with quarantined persons. Inspecting personnel should wear gowns, coveralls, caps, masks, and rubber gloves. (See AR 40–205 and 40–210.)

(3) *Immunization.*—All military personnel under serious threat of exposure to epidemics of human plague should be immunized with plague vaccine. (See AR 40–210 and sec. VII.) The initial vaccination consists of 2 subcutaneous injections of plague vaccine with an interval of 7 to 10 days between injections. The first dose is 0.5 cc and the second dose 1.0 cc of vaccine. Additional 1.0-cc doses of plague vaccine may be administered whenever, in the opinion of the surgeon, additional stimulation of immunity is necessary.

h. Sylvatic plague.—(1) *General.*—This is flea-borne plague, caused by *P. pestis.* It occurs as an epizootic disease among wild rodents (ground squirrels, marmots, rabbits, and others) which do not ordinarily live in close association with man. This type of plague may be the source of sporadic human infections and may be introduced into the domestic rat population of cities, following which epidemics of rat plague and human plague may occur.

(2) *Control measures.*—(*a*) Avoid unnecessary contact with wild rodents.

(*b*) In endemic rural areas control wild rodent population by shooting, poisoning, gassing, and filling of burrows.

71.3. Relapsing fever—louse-borne.—*a. Etiologic agent.*—Variously designated spirochaetes as, for example, *Borrelia recurrentis* or *Spirochaeta obermeieri* in European relapsing fevers.

b. Geographical distribution.—This disease has a cosmopolitan distribution and is likely to occur in epidemic form wherever lousiness prevails. Epidemics in the last 25 years have occurred in eastern Europe, northern Asia, India, China, western Asia, North Africa, and Equatorial Africa. Lesser outbreaks have been reported from Peru, Central America, French Indo-China, and Japan. Chief foci since 1934: Africa and Russia.

c. Transmission.—From man to man by *Pediculous humanus* (head and body louse) and possibly by *Phthirus pubis* (crab louse). Crushing of louse by scratching liberates the microorganisms which enter the body through abraded skin.

d. Recognition.—Onset is sudden with headache, generalized pains, chills, and fever of 104° F. to 105° F. The temperature usually remains high 4 to 6 days and then subsides abruptly by crisis. Vomiting, splenomegaly, leucocytosis, and albuminuria may occur during the acute febrile stage. An afebrile period of 4 to 8 days is followed by a relapse with similar but milder symptoms. Several relapses may occur unless treatment is instituted. Mortality varies from 2 to 50 percent.

e. Specific diagnosis.—Demonstration of *Borrelia* in the blood during febrile periods by darkfield examination, by Giemsa-stained thin or thick films, or by mouse inoculation with the blood of the patient, in which case the organism appears in the blood of the mouse 24 to 48 hours later.

f. Treatment.—Inject 0.6 gram of neoarsphenamine intravenously. One dose usually cures, but two or three doses may be necessary. This therapy is most effective at beginning of the initial attack or of a relapse; it is dangerous at the time of crisis or just after it, since the patient may collapse. Occasional cases are resistant to this drug. Good nursing during the fever is important. Mapharsen has been used in a few cases with good results and there is reason to believe that it may replace neoarsphenamine. It should be used in doses of 0.04 to 0.06 gram intravenously.

g. Prevention.—Apply measures for eradication of lice (see louse-borne typhus). No method of immunization has been developed.

71.4. Relapsing fever—tick-borne.—*a. Etiologic agent.*—Spirochaetes, variously designated as *Borrelia, Spirilla, Spirochaeta, Spironema,* or *Treponema,* with such so-called geographical strains as, for instance, *Borrelia duttoni* in the relapsing fever of tropical Africa.

b. Geographical distribution.—Patchy distribution; does not tend to spread; generally remains localized in areas corresponding to distribution of the tick vector. It is a "place" or "house" disease. Foci in Africa, Asia, Europe, Canada, United States (western and southwestern States), Central America, and South America.

c. Transmission.—From lower animal reservoir hosts to man by soft ticks of the genus *Ornithodorus.* The reservoir hosts include many kinds of rodents, monkeys, possibly bats, and other mammals. The ticks are found in native huts and in the burrows and nests of wild rodents. In the United States they may be found in the cabins and cottages of the western recreational areas and in the caves of the Colorado River Valley. In human habitations they have the feeding habits of bedbugs. Attachment to host for feeding is usually brief (12 to 15 minutes) but may continue for 1 hour.

d. Recognition.—Clinically similar to the louse-borne type except that—

(1) Cerebro-spinal symptoms are more common in some areas.

(2) Paroxysms are more intense but of shorter duration, 2 to 4 days.

(3) Greater number of relapses occur, usually more than four.

(4) Mortality is lower (average 5 percent).

e. Specific diagnosis.—Same as for the louse-borne type.

f. Treatment.—Same as for the louse-borne type. Some workers report that tick-borne relapsing fever in certain areas is very resistant to arsenicals.

g. Prevention.—(1) Avoid sleeping in native huts and cabins in endemic areas.

(2) Avoid caves inhabited by rodents, bats, or ticks.

(3) Avoid soiling hands with blood of rodents in endemic areas.

(4) In endemic regions, sleeping quarters should be so constructed as to discourage entrance or nesting of rodents beneath floors or in walls and the harborage of ticks in cracks in floors or walls.

(5) Cleanliness of barracks, tents, and other quarters should be maintained, to prevent the accumulation of dust and debris which might give harborage to ticks and other vermin.

(6) Instruct personnel to examine day and night clothing and bedding for ticks after possible exposure.

71.5. Rickettsial diseases—classification.—The rickettsial diseases discussed in the following pages are epidemic and endemic typhus, Rocky Mountain spotted fever, tsutsugamushi disease, and "Q" and trench fevers. These may be outlined as follows:

Typhus fevers	Epidemic louse-borne.	
	Endemic flea-borne.	
Tick-borne spotted fevers	Rocky Mountain spotted fever	No primary sore.
	Exanthematic Brazilian typhus	No primary sore.
	Fievre boutonneuse	Primary sore.
	S. African tick-bite fever	Primary sore.
Mite-borne fever	Tsutsugamushi fever (Japan, China, Malaya, and Sumatra).	
Miscellaneous group	Tick-borne "Q" fever in Australia.	
	Louse-borne trench fever of the first World War.	

71.6. Epidemic typhus (louse-borne or European typhus).—*a. Etiologic agent.—Rickettsia prowazeki.*

b. Geographical distribution.—Chiefly in Europe, North Africa, Asia, and in the higher altitudes of Central and South America. Not present in the United States or insular possessions.

c. Transmission.—The reservoir of the disease is man. Transmission is from man to man by the louse, *Pediculus humanus.* It is chiefly a disease of winter and spring, and affects especially impoverished, overcrowded, and dirty peoples.

d. Recognition.—Incubation period 6 to 14 days. Onset usually sudden, with chills, fever, general pains, and severe headache. The fever continues for about 2 weeks. A macular papular eruption appears on the fourth to the sixth day which may become petechial. Typically the rash appears first on chest or abdomen, spreads to back, arms, legs, and rarely in severe cases, to palms, soles, and face.

Mental dullness is common, especially during the second week. Case fatality rate 20 to 60 percent. One attack confers immunity which is not always permanent.

e. Specific diagnosis.—Agglutination of *B. proteus* OX19 (Weil-Felix agglutination) occurs during the febrile period of the disease, increases in titer during early convalescence, and disappears in late convalescence. The reaction is of diagnostic importance when a rising titer occurs. Single tests are of little diagnostic value, and a differentiation between typhus and Rocky Mountain spotted fever cannot be made by this test alone. It is important to use to "O" or nonmotile variant *B. proteus.* Of great importance is specific rickettsial complement fixation. This test becomes positive about the eighth day and increases in titer, remaining positive for years after the attack. By employing specific antigens, epidemic and endemic typhus may be distinguished. The complement fixation test is performed routinely at the Army Medical School, Washington, D. C. Guinea pigs inoculated with infective blood develop a characteristic temperature curve after a period of incubation. One attack confers an immunity to a second inoculation.

f. Treatment.—Symptomatic. Absolute rest in bed, with good nursing care. Maintenance of fluids by mouth, rectum, or hypodermoclysis. Relief of constipation by means of enemas or mild laxatives. Tepid sponges for lowering fever. Relief of headache by aspirin or codeine; morphine may be necessary. No specific treatment of proven value has been developed. A hyperimmune rabbit serum is under study, but is not yet generally available. The sulfonamide drugs have been tried without benefit.

g. Prevention (see AR 40-205, 40-210, and 615-250).—(1) *Delousing clothing.*—(*a*) *Methyl bromide.*—Recent studies indicate that the best delousing of clothing and equipment can be obtained by the use of QM issue methyl bromide, either in portable and demountable forced air circulation gas chambers made of plywood, or in special QM issue bags made impervious by impregnation with ethyl cellulose. Clothing and equipment are not injured by this method. The fumigant, methyl bromide, is slightly more toxic than carbon tetrachloride, but when used in the prescribed manner it can be safely handled and gas masks are not required by the personnel involved. The gas has great powers of penetration and when the proper dosage is used it will, in 30 minutes, destroy all insects or insect eggs, even if they are on clothing in the center of a filled barracks bag. Clothing fumigated in this way can be safely reissued to the owners within 1 hour, or by the time the concurrent body delousing and bathing procedures have been finished.

(*b*) *Steam.*—Very efficient. Suitable apparatus may easily be improvised. Causes shrinkage of woolen goods and wrinkling of clothing; damages leather, felt, and webbing. Pressure sterilizers are employed to best advantage in permanent delousing stations; emergency stations may employ a method utilizing flowing steam, such as the Serbian barrel.

(*c*) *Dry heat.*—135° F. for 5 minutes. Does not injure leather, metal, or webbing, and does not wrinkle clothing. Difficult to secure penetration of dry heat into clothing.

(*d*) *Hot water.*—Is not suitable as it causes shrinkage of woolen goods and damages felt and webbing.

(*e*) *Insecticidal powder.*—The approved QM issue insecticidal powder may be dusted into the seams of clothing every 7 to 10 days, not only as a delousing measure but as a prophylactic against infestation.

(2) *Delousing persons.*—(*a*) The delousing of the body surface may be accomplished by clipping or shaving of the hair and the application of vinegar or 15

percent acetic acid to loosen eggs. Then shampoo vigorously with hot soapy water containing 25 percent kerosene and remove any retained nits or eggs by combing with a fine comb.

(*b*) When available, the following insecticidal preparation may be used:

Pyrethrins	2.5
IN 930	20
Castor oil	20
Isopropyl alcohol	810
Water, q. s. ad	1,000

Apply to hairy parts 15 minutes before bathing.

(*c*) Another satisfactory method of destroying head and crab lice is to apply 8 cc of one of the following insecticide solutions 24 to 48 hours prior to delousing of clothing and bathing:

Lethane 384 special	500
Mineral oil	500
or	
Lauryl thiocyanate	250
Mineral oil	750

(*d*) In the absence of these special preparations, dust the hairy parts of the body liberally with QM issue insecticidal powder and rub in. Follow in 24 to 48 hours by soapy bath and combing out of retained nits.

(3) *Control patients.*—All patients and contacts should be carefully deloused. This includes destruction of lice and eggs in the clothing as well as on the body. Contacts should be revaccinated with typhus vaccine unless so vaccinated within 2 months. The clothing of typhus patients should be steam sterilized to destroy all infective materials.

(4) *Vaccination.*—A vaccine prepared with killed rickettsiae has been provided. At present this vaccine is administered by giving three injections of 1 cc each, subcutaneously, at approximately weekly intervals. A stimulating dose of 1 cc should be administered every 4 to 6 months as long as serious danger of infection exists. Additional injections may be given whenever, in the opinion of the surgeon, additional stimulation of immunity is deemed advisable. For further details see section VII.

71.7. Endemic typhus (murine or flea-borne typhus).—*a. Etiologic agent.*—*Rickettsia mooseri.*

b. Geographical distribution.—Endemic typhus has been found in all regions where the epidemic disease occurs. However, endemic typhus may occur alone, as it does in the southern part of the United States.

c. Transmission.—The reservoir of the disease is in rats and the disease is transmitted to man by rat fleas, particularly *Xenopsylla cheopsis.* The disease apparently is transmissible from man to man by the body louse. Epidemics of murine typhus may thus occur in a lousy population although the disease is not ordinarily associated with lousiness or poverty. Endemic typhus occurs chiefly in late summer and fall in persons having contact with rats, or living or working on premises harboring rats, especially grocery stores, food or grain warehouses, or restaurants.

d. Recognition.—Incubation period 6 to 14 days. Clinically similar to epidemic typhus, except that the course is milder and the rash may be less profuse and

of shorter duration. Case fatality rate about 5 percent. The chief differential diagnosis is from Rocky Mountain spotted fever.

e. Specific diagnosis.—Weil-Felix agglutination OX19 same as for epidemic typhus. Specific rickettsial complement fixation tests are of great assistance in differentiating endemic from epidemic typhus, as indicated above. Guinea pigs inoculated with infected blood develop a characteristic temperature curve after a period of incubation. In the guinea pig this disease is usually characterized by scrotal swelling and the appearance of "Neill-Mooser bodies" in scrapings from the inflamed tunica vaginalis. "Neill-Mooser bodies" are enlarged serosal cells filled with intracytoplasmic rickettsiae. Rocky Mountain spotted fever is differentiated as follows:

	Endemic typhus	Rocky Mountain spotted fever
History	Exposure to rat fleas	Exposure to ticks.
Fever	Two weeks	Three weeks.
Rash	First on body; spreads to extremities.	First on extremities; spreads to body.
	Palms and soles usually free	Palms and soles usually affected.
	Neck and face rarely involved	Neck and face commonly involved.
	Usually fades before defervescence	Usually persists until defervescence.
Pulse	100 or less	100 to 130.
Leucocytes	Normal limits	10,000 to 18,000.
Weil-Felix (OX19)	Second week	Second or third week.
Complement fixation	Positive with typhus antigen	Positive with spotted fever antigen.

f. Treatment.—Same as for epidemic typhus.

g. Prevention.—(1) Elimination of rats by ratproofing, trapping, and poisoning. Special care should be directed to proper protection and disposal of garbage to prevent access by rats. (See AR 40-205 and 40-210.)

(2) The isolation of patients is not necessary in the absence of body lice.

(3) *Vaccination.*—The incidence of the disease is too low to warrant general vaccination. This procedure is not considered in places where the rat population can be controlled.

71.8. Rocky Mountain spotted fever (tick fever, exanthematous tick fever, tick typhus, Brazilian spotted fever (Saõ Paulo typhus)).—Immunologically related diseases: Boutonneuse fever (Mediterranean littoral), Kenya typhus (East Africa), Tobia fever (Colombia), and South African tickbite fever.

a. Etiologic agent.—*Rickettsia rickettsii* (*Dermacentroxenus rickettsi*).

b. Geographical distribution.—Reported from nearly all States of the Union. Also present in British Columbia and Alberta and in Brazil and Colombia. About 1,000 cases are recognized annually in the United States.

c. Transmission.—Reservoir hosts probably some species of rodents or other animals. Wood ticks (*Dermacentor andersoni*) in the western States and dog ticks (*Dermacentor variabilis*) in the eastern States transmit the disease to man during the process of feeding. Other species of ticks are the vectors in other countries. Seasonal occurrence in northwestern States principally during spring and early summer; in the eastern and southern States throughout the summer. Rarely cases occur as late as December.

d. Recognition.—There is usually a history of exposure to tick bite. Incubation period 2 to 14 days, usually about 1 week. General features like those of epidemic typhus, but usually more severe. Duration of fever usually about 3 weeks. Defervescence usually by rapid lysis. Meningismus is not uncommon. Delirium, convulsions, and coma may develop in severe cases. There is usually a leucocytosis of 10,000 to 18,000. Eruption similar to typhus but more intense and usually becomes purpuric. Appears first about wrists and ankles, rapidly spreads over body, but less intense on trunk. Usually involves palms and soles. Persists until defervescence. Rash is followed by brownish stain and often by desquamation. Case fatality rate about 25 percent.

e. Specific diagnosis.—Weil-Felix agglutination OX19 is similar to that described for epidemic typhus. A specific rickettsial complement fixation reaction may be obtained beginning usually about the tenth day and remaining positive for years after convalescence. By means of this test Rocky Mountain spotted fever can be distinguished from either epidemic or endemic typhus.

f. Treatment.—Same as for typhus. A hyperimmune rabbit serum has recently been developed. It is only of value when administered during the early stages of the disease.

g. Prevention.—Adult ticks are active from February through August. There is some variation with the climatic conditions and the section of the country. The disease is transmitted solely through tick contact. In order that the tick bite may transmit the disease, the tick must remain attached for several hours.

(1) *Tick control.*—(*a*) Avoid tick infested area if possible. Common sites for ticks: tall grass, especially along animal runs, bushes, along the edge of wooded areas, and along streams.

(*b*) Remove ticks from clothing and body at least once a day, best done immediately after coming from areas where ticks may be present and as a routine measure on retiring. Ticks are likely to attach to the skin on the hairy portions of the body or where their progress is stopped by constricting clothing, such as the belt. Ticks should be removed gently to avoid breaking off the mouth parts. This can be done with tweezers or with cotton saturated with iodine. The tick should not be crushed with the bare fingers. Paint the site of the tick bite with tincture of iodine. Wash hands thoroughly after handling ticks, especialy after removing engorged ticks from animals. Dogs should be deticked at frequent intervals.

(2) *Vaccination.*—Vaccine is prepared at the Rocky Mountain Laboratory, U. S. Public Health Service, Hamilton, Montana. It may be procured from that laboratory or from the National Institute of Health, Bethesda, Maryland, when authorized by The Surgeon General. Limited amounts may also be obtained from State health officers. The vaccine may be given in two or three doses of 1 or 2 cc each, 1 week apart. Immunity is not absolute, but is apparently sufficient to protect against death should spotted fever be acquired within a year subsequent to vaccination. The vaccine is probably of no value after an infecting tick contact. It is valueless in treatment. As a relatively small number of cases occur each year, mass vaccination is not recommended in the Army.

71.9. Tsutsugamushi disease (Japanese river fever).—*a. Etiologic agent.*—*Rickettsia orientalis* (*R. tsutsugamushi, R. nipponica*).

b. Geographical distribution.—Japan, Formosa, Indo-China, Malaya, and probably India, Korea, Sumatra, the Philippine Islands, and Australia (Queensland).

c. Transmission.—The disease occurs in Japan in summer and fall months in low-lying sections that are subject to river floods. Field mice are reservoir hosts. The infection is transmitted to man in Japan by the larval form of the

mite, *Trombicula akamushi*, and by other species of mites elsewhere. The infection is transmitted to the next generation of mites through the egg.

d. Recognition.—Incubation period is about 1 week. The fever lasts from 2 to 3 weeks. A macular papular rash appears on the second to seventh day, first on trunk and arms, and rapidly becomes generalized. There is usually a local ulcer at the site of the infecting bite and the regional lymph nodes become enlarged and tender. Later there may be a general lymph node involvement. Leucopenia is usually present.

e. Specific diagnosis.—Agglutinins for *B. proteus* OXK occur but not for OX19. Case fatality rate 40 to 55 percent in Japan, 4 percent in Sumatra. Immunity is confered by an attack, although second attacks have been noted.

f. Treatment.—Same as for typhus.

g. Prevention.—Avoid unnecessary exposure in infected areas during summer and fall. Bathing in hot water with soap immediately after exposure and change of clothing may prevent infection. Insect repellents such as dimethylthallate, QM issue insecticide powder, or equal parts of flowers of sulfur and talcum powder, may be of value. No vaccine against the disease has been developed.

71.10. "Q" fever.—*a. Etiologic agent.—Rickettsia burneti (R. diaporica).*

b. Geographical distribution.—Recognized as a natural infection in humans in Australia. Cases in Washington, D. C., were probably laboratory infections. The disease may be occurring unrecognized in other regions. Specific antibodies in human blood have been demonstrated in Idaho, Montana, Wyoming, Nebraska, Nevada, Arizona, and Washington State. In Montana the tick *Dermacentor andersoni* has been found naturally infected.

c. Transmission.—Australian cases associated with handling of livestock. The bandicoot, a marsupial, is apparently a reservoir host. Transmission to a man by a tick *Hemaphysalis humerosa* has been suggested. Two types of the disease may exist, one transmitted by a tick, the other through the respiratory tract, possibly by dust containing the dried excreta of infected animals or anthropods.

d. Recognition.—Incubation period in the Australian cases is thought to be 10 days to 1 month. Onset acute, with fever, prostration, and headache, accompanied by chills and sweats. Duration of acute stage from a few days to 3 weeks. White blood cells are within normal limits. There is no rash. In most of the laboratory cases in Washington, vague chest pains, unproductive cough, and rales were present. X-ray examination of the chest showed a soft infiltrative lesion which began centrally and spread outward. Clinically these cases could not be differentiated from the atypical pneumonias reported frequently during the past few years. Convalescence may be prolonged. The fatality rate is low.

e. Specific diagnosis.—Agglutinins are not produced for any of the strains of Proteus X so far employed. In the second week complement-fixing antibodies against *R. burneti* appear in the blood serum. The organism can be isolated in guinea pigs by inoculation of blood taken during the febrile period, and protective antibodies can be demonstrated in recovered cases.

f. Treatment.—Symptomatic. None of the sulfonamide compounds has been found to be of any benefit.

g. Prevention.—Further knowledge is required before control measures can be evolved.

71.11. Trench fever (Wolhynian fever, 5-day fever).—*a. Probable etiologic agent.—Rickettsia quintana.*

b. Geographical distribution.—Trench fever appeared during the first World War. It became epidemic in nearly all the armies of Europe and later spread to Mesopotamia. Only a few cases have been recognized since the last war.

c. Transmission.—As demonstrated by human experiments, the infection may be transmitted by the body louse. Recovered cases of trench fever may remain infective to lice for months. The virus in the dried louse excreta remains virulent for at least 4 months. It also retains its virulence in the dried urine of patients. The disease has never been transmitted to laboratory animals. The infecting agent is present in the blood of human cases, as has been shown by injecting the patient's blood into voluteers, but rickettsia have not been demonstrated in humans.

d. Recognition.—Incubation period 1 to 3 weeks. Onset sudden, with prostration, and severe pain in the muscles and bones, particularly the tibia, radius, and ulna. Frontal headache and ocular pains may be severe. Congestion of the conjunctivae is common. The spleen is generally enlarged. In the majority of cases a rash develops on the first or second day of fever, consisting of small macules mainly on the trunk. It does not become petechial. Sweats are common. The leucocyte count is usually between 10,000 and 12,000. The initial bout of fever lasts about 5 days. Relapses occur at intervals of 5 to 6 days or even longer. Three to five relapses may occur. Prostration is pronounced and convalescence prolonged. The disease is not fatal. Immunity is apparently not established by one attack.

e. Specific diagnosis.—None. The reaction of the serum to the Weil-Felix agglutination test is not known.

f. Treatment.—Entirely symptomatic.

g. Prevention.—Delousing (see epidemic typhus). The virus is present in the urine and saliva of patients. Therefore, attendants should be protected against contact infection, body discharges should be disinfected, and clothing and bedding should be disinfected with steam or by disinfectant solution, such as cresol, 2 to 3 percent. (See AR 40–205 and 40–210.)

71.12. Schistosomiasis.—There are three schistosomes (blood flukes) which cause serious diseases in man.

a. Schistosoma haemtobium, producing mainly genito-urinary symptoms.

b. Schistosoma mansoni, producing mainly intestinal symptoms.

c. Schistosoma japonicum, producing mainly hepatic symptoms.

71.13. Schistosomiasis produced by *S. haematobium* (Bilharziasis, endemic haematuria).—*a. Geographical distribution.*—Especially prevalent in the Nile Valley; also in other areas of Africa and in parts of Asia and Europe bordering on the Mediterranean.

b. Transmission.—The adult worms live and lay their eggs in the veins around the bladder. The eggs escape in the urine, rarely in the feces. When deposited in fresh water, the egg hatches and the embryo (miracidium) enters a snail (genus *Bulinus*, *Physopsis*, and others). The snails may be found in fresh water with slightly alkaline reaction, usually containing aquatic vegetation, and occasionally in brackish water. They may inhabit slow-moving streams, irrigation ditches, limestone sinks, reservoirs, and small pools after flooding of streams. Larvae (cercariae) emerge from the infected snails and enter the water, infecting man or animals by penetration of the skin. Thus the disease may be contracted while bathing, wading, working, or washing clothes in infected water, or by drinking such water and possibly through the use of a polluted public water supply.

c. Recognition.—(1) *Early.*—Papular dermatitis at site of penetration of the cercariae.

(2) *Four to eight weeks after infection.*—Fever, giant urticaria, eosinophilia, and hematuria.

(3) *Late.*—Vesical ulcers, urinary calculus, and papillomata, urinary fistulae, splenomegaly, and cirrhosis of liver.

d. Specific diagnosis.—Presence of characteristic terminal-spined ova in urine, rarely in feces.

e. Treatment.—(1) Fuadin (neoantimosan) intramuscularly; 1.5 cc, 3.5 cc, and 5.0 cc on successive days, then 5.0 cc on alternate days to a total of 10 doses. Toxic symptoms: vomiting, joint pains (rare). If toxic symptoms appear, reduce dosage. If eggs containing living embryos are found after completion of treatment, repeat treatment after a rest of 1 week. (See (2) below.)

(2) If a satisfactory response is not attained with these courses of fuadin, use potassium antimony tartrate (USP), 2 percent freshly prepared solution intravenously on alternate days. Initial dose 2.5 cc (0.05 gram); increase each subsequent dose by 1.25 cc until 7.5 cc are being given; continue until a total of 12 to 15 doses have been given. Administer 2 to 3 hours after a light meal and have patient lie down for 1 hour after administration. If toxic symptoms appear, reduce dosage.

(*a*) *Toxic symptoms.*—Coughing immediately after administration (not important); nausea, vomiting, dizziness, and collapse.

(*b*) *Contraindications to antimony preparations.*—Nephrities, jaundice, or severe liver disease.

(3) *Diet.*—The diet should liberally supply all the essential nutrient substances. If evidence of hepatic disease exists, the diet should be high in carbohydrates and low in fats. Milk, eggs, and cheese are permissible. The supplementation of the diet with vitamin A and vitamin B complex is advised.

(4) *Criterion of cure.*—Cessation of passage of eggs containing live embryos. If cure is not obtained, repeat treatment after 1 month.

f. Prevention.—(1) Avoid entering or using unpurified fresh water in endemic areas. The apparent absence of snails from such water does not guarantee that the water does not contain cercariae.

(2) In case of accidental or unavoidable entrance into water containing cercariae, immediate and complete bathing with soap and pure water may prevent infection.

(3) If it is necessary to provide bathing facilities in a natural body of fresh water in an endemic area, a preliminary survey of the surrounding population should be made to exclude the presence of schistosomiasis. Steep banks and wave action diminish the probability of the presence of snails. Copper sulfate in a dilution of 1/200,000 will kill cercariae and snails.

71.14. Schistosomiasis produced by *S. mansoni* (intestinal schistosomiasis).—*a. Geographical distribution.*—In the Nile Valley and in many other parts of Africa, coexisting with *S. haematobium;* also in South America (Brazil, Dutch Guiana, and Venezuela) and West Indies (Puerto Rico, Vieques, the Lesser Antilles, including St. Lucia, Antigua, St. Kitts, Nevis, Montserrat, Martinique, Guadeloupe, and St. Martin).

b. Transmission.—The adult worms live and lay their eggs in the veins around the colon and rectum. The eggs are discharged in the feces, rarely in the urine; some are carried to the liver. Otherwise the life cycle closely resembles that of

S. haematobium except that the snail hosts belong to the genera *Planorbis* or *Australorbis.*

c. Recognition.—Same as in *S. haematobium* infections except that the colon and rectum, instead of the bladder, are the principal organs involved, and the late clinical findings are bloody stools, rectal polyps and fistulae, and prolapse of the rectum. Hepatic cirrhosis and splenomegaly are more pronounced than in *S. haematobium* infections.

d. Specific diagnosis.—Presence of characteristic lateral-spined ova in feces, rarely in urine.

e. Treatment and prevention.—Essentially the same as for *S. haematobium* infection.

71.15. Schistosomiasis produced by *S. japonicum* (oriental schistosomiasis, Katayama disease).

a. Geographical distribution.—Occurs only in the Far East. Common in China, particularly in the Yangtse Valley, also along China Coast from Shanghai to Canton, and in Yunnan Province. Less common in Japan, Formosa, and the Philippines (Leyte and Samar Islands); possibly in Celebes and Shan States of Burma.

b. Transmission.—The adult worms live and lay their eggs in the veins of the small intestines. The eggs are discharged in the feces; many are carried to the liver. Otherwise the life cycle closely resembles that of *S. haematobium* except that the snail hosts belong to the genera *Oncomelania* and *Katayama.*

c. Recognition.—Same as in *S. mansoni* infections except that rectal and anal lesions are less common, while hepatic cirrhosis and splenomegaly are very common and are the essential late symptoms.

d. Specific diagnosis.—Presence of characteristic spineless ova (rudimentary lateral spines sometimes discernible) in feces, *not* in urine.

e. Treatment and prevention.—Essentially the same as *S. haematobium.*

71.16. African trypanosomiasis (sleeping sickness).—*a. Etiologic agent.*—*Trypanosoma gambiense* and *Trypanosoma rhodesiense.*

b. Geographical distribution.—Endemic throughout most of tropical Africa, the boundaries roughly extending from Senegal east to Bahr el Ghazal Province of the Southern Sudan, thence south through the lake country of East Africa into Portuguese East Africa and thence west to Angola.

c. Transmission.—The trypanosomes are blood parasites of man and some wild domestic animals which may serve as reservoir hosts, transmitted by various species of tsetse flies of the genus *Glossina,* especially *G. palpalis, G. tachinoides, G. morsitans,* and *G. swynnertoni.* Under favorable conditions the fly becomes infective in from 18 to 34 days after ingestion of infected blood. Man is infected by the introduction of trypanosomes by the bite of the infected tsetse fly. These flies bite only in daylight. Under epidemic conditions simple mechanical transmission by other biting flies and insects may possibly occur.

d. Recognition.—(1) Bite of an infected fly often produces more local reaction than that of an uninfected fly. After an incubation period usually varying from 10 days to 3 weeks, irregular remittent fever, rapid pulse, deep hyperesthesia and asthenia appear, often accompanied by headache and neuralgic pains. Delirium and acute fever may occur early in *T. rhodesiense* infections. In white individuals a characteristic erythematous skin rash on the trunk or thighs is common. The individual lesions are round or oval patches, pinkish in color with a clear center. Pruritus and dryness of the skin are common. The liver

and spleen may be enlarged and the Wassermann reaction may be positive. Enlarged lymph glands, especially those of the posterior triangle of the neck, are an important diagnostic sign. Glands are discrete, at first soft and elastic, later shrinking and becoming fibrotic. Enlargement persists usually from the second to the sixth month of the disease.

(2) The cerebral stage (sleeping sickness) may be ushered in by tremor of tongue and fingers, headache, delusions, hysteria, mania, and the other signs of meningo-encephalitis and meningo-myelitis. Somnolence is the characteristic cerebral symptom. Late cases show marked wasting from starvation.

e. Specific diagnosis.—Early diagnosis depends upon demonstration of trypanosomes in lymph nodes, blood, or spinal fluid. Aspiration of fluid from lymph node puncture with hypodermic needle and syringe is most likely to reveal trypanosomes. Fresh unstained coverslip preparations reveal organisms by their active movement even if they are few in number. Check with smear stained with Wright's stain. Blood smears are less useful but may show trypanosomes in fresh coverslip preparations, thin stained smears, or better in thick blood drop preparations as for malaria. Centrifuged citrated blood may reveal light infections. ***Spinal fluid should be examined in all early cases for increased cell count and globulin, and should be centrifuged for trypanosomes as evidence of cerebral involvement.***

f. Treatment.—All cases should be treated in a well-screened hospital.

(1) *Early cases.*—(*a*) Tryparsamide, dissolved in 10 cc of distilled water (salt solution must *not* be used): 15 weekly intravenous injections. Individual dose 0.04 to 0.05 gram per kilo of body weight. Initial adult dose 1.0 to 1.5 grams, subsequent doses 2.0 to 3.0 grams. Vigorous complete treatment is essential to avoid production of arsenic-fast strain. Toxic effect: Danger of tryparsamide is optic atrophy. If photophobia, lacrimation, ocular pain, or dimness of vision occur, reduce dose and lengthen interval to 10 days.

(*b*) Naphuride, Winthrop (Bayer 205, antrypol) intravenously in 10 cc of distilled water every 4 days for four to six doses. Initial adult dose 0.3 to 0.5 gram, subsequent doses 1.0 gram. Toxic effect: Naphuride is a kidney irritant. Urine should be free from albumin before treatment and must be examined after each treatment and the drug discontinued if increasing albuminuria occurs.

(*c*) Lumbar puncture should be performed before and after completion of therapy in every case, and *every* case should be kept under observation for at least 2 years. Centrifuged citrated blood should be examined at completion of therapy, monthly for the next 3 months, and then every 6 months for at least 2 years.

(2) *Late cases.*—Tryparsamide is the only drug of value in the treatment of cases with involvement of the central nervous system. Dosage same as for early cases. The drug should *not* be given intrathecally. Initial dose should be half the standard dose because of dangers analogous to those in arsenical treatment of cerebro-spinal syphilis. The spinal fluid affords a guide to the efficacy of the treatment. First course of treatment should be 20 weekly injections. Repeat course of treatment after interval of 1 to 3 months. The blood should be examined periodically as previously indicated. The spinal fluid should be examined before and after the second course of treatment and again 6 months and 1 year later. Late cases should be followed for at least 3 years.

g. Prevention.—(1) Control and examination of fixed personnel, especially natives, in vicinity of station. Infected personnel should be held and adequately

treated before being permitted to enter another area where tsetse flies are present.

(2) *Protective clothing.*—The tsetse fly frequently bites on the legs; high shoes with leggings or mosquito boots should be worn. Shorts should not be worn.

(3) *Camp siting.*—Camps should be located on high ground away from native villages, streams, and forests. Buildings should be screened. Brush along streams near stations should be cleared for a distance of 50 yards from the water's edge. Brush about camps should be cleared for a distance of 100 yards. Motor vehicles should be inspected for presence of tsetse flies. Fishing and hunting are dangerous in endemic areas.

71.17. American trypanosomiasis (Chagas' disease).—*a. Etiologic agent.*—*Trypanosoma cruzi.*

b. Geographical distribution.—Brazil, Argentina, Uruguay, Venezuela, Peru, Panama, San Salvador, Guatemala, and Mexico. Present in certain wild rodents in California and in reduviid bugs in California, Arizona, New Mexico, and Texas.

c. Transmission.—From man to man, and from animal reservoir (armadillo, opossum, and certain rodents) to man by contamination of bite of cone-nosed reduviid bug, especially *Triatoma megista* and *T. infestans* by feces of the infected insect. These bugs habitually defecate while feeding and thus contaminate the bite with their feces in which may be present the infective trypanosome.

d. Recognition.—A relatively uncommon disease occurring particularly in children and characterized by invasion and destruction of the endothelial and other cells, notably cardiac and striated muscle, by the *Leishmania* form of the parasite. The acute stage is characterized by fever, facial edema, adenitis, and anemia. Symptoms of the chronic stage depend upon the localization of the parasite especially in the heart, central nervous system, thyroid, or suprarenal glands. Pathogenicity for adults doubtful.

e. Specific diagnosis.—Diagnosis is based upon demonstration of the trypanosome form *T. cruzi* in blood or the *Leishmania* form in the tissues, or demonstration of the trypanosome in animals (guinea pigs) inoculated with patient's blood.

f. Treatment.—Symptomatic.

g. Prevention.—Avoidance of adobe and thatched huts of natives, where *Triatoma* commonly hides.

71.18. Typhoid fever.—*a. Etiologic agent.*—*Bacterium typhosum* (*Eberthella typhosa*).

b. Geographical distribution.—World-wide. (The indigenous races of India, Africa, and elsewhere are not immune.)

c. Transmission.—Typhoid fever is spread principally by food and water contaminated by urine or feces of patients and carriers. Flies, fingers, or fomites may convey infective material.

d. Recognition.—The incubation period varies from 8 to 14 days, usually 10. The recognition of typhoid fever in the Tropics, where malaria and the dysenteries are complicating factors, and particularly its recognition in individuals who have been vaccinated, is not always easy. Clinically there may be the typical constant or remittent fever with slow pulse, enlarged spleen, "rose" spots, tympanites, muttering delirium, and leucopenia. Constipation rather than

diarrhea is the rule in tropical typhoid. The rash is often absent or difficult to differentiate from the many minor infectious and other skin lesions always present in hot climates. In patients with malaria the onset of typhoid may be abrupt with a rigor. The temperature usually becomes continuous but may deviate from that typical of typhoid.

e. Complications.—The usual complications of intestinal perforation and hemorrhage must be kept in mind. Perforation calls for immediate surgical intervention. Localized purulent foci of infection may develop in gall bladder, bone marrow, periosteum, or cartilage, and these also require surgical treatment. Bed sores, phlebitis, myocarditis, and pneumonia may be complications. In tropical areas the attack is generally more severe and more prolonged than in temperate zones. Death may occur as early as the sixth day.

f. Specific diagnosis.—The only certain test, in previously vaccinated individuals, is the isolation of *B. typhosum* in cultures. Blood culture is usually positive during the first week or 10 days, sometimes longer, and sometimes during a relapse. After 2 weeks of fever, positive cultures are more likely to be obtained from feces and, less often, from urine. It must be remembered that a typhoid *carrier* in the Tropics may be suffering from malaria fever although his stool or urine gives a positive culture for typhoid. Blood serum agglutination test (Widal reaction) usually becomes positive, 1 to 160 or more, during the second week and usually remains positive through convalescence or longer. In previously vaccinated persons positive tests are not a certain indication of typhoid fever unless a rising titer can be demonstrated.

g. Treatment.—(1) *Specific.*—There is no specific chemotherapy or serum treatment. In a few cases good results have been reported with sulfaguanadine 3.0 grams (45 grains) three times a day. But caution is indicated because of the danger that the usual leucopenia of typhoid fever may develop under sulfa drug therapy into an agranulocytosis. There is not yet sufficient evidence as to the efficacy of chemotherapy.

(2) *General.*—Complete rest in bed until convalescence is well established. Patients should be moved as little as possible. Careful nursing is most important. Mouth must be kept clean. Purgatives should be avoided, using enemas instead. Hot stupes to abdomen and use of rectal tube may relieve tympanites. Alcohol rubs three or four times a day with tepid sponge baths should be given, especially when the temperature is above 104° F.

(3) *Diet.*—Food should be given frequently in small amounts, totaling 3,000 to 4,000 calories per day, consisting of liquids and soft foods with little residue. Milk, cream, butter, eggs, soft cereals, gelatin, and pureed cooked vegetables and fruits, especially orange juice, can form the basis of the diet. They should be fortified with carbohydrates, particularly lactose, with satisfactory provision for vitamins of the vitamin B complex. This vitamin provision should include at least 3 milligrams of thiamin, 4.5 milligrams of riboflavin, and 30 milligrams of nicotinic acid amide. Three of the standard multivitamin capsules daily will supply these amounts. For the balance of the vitamins of the vitamin B complex, administration daily of 3 cc of crude liver extract is suggested. Fluid intake should be 3,000 cc or more per day.

h. Prevention.—(1) Water and milk supplies must be carefully supervised, as prescribed in AR 40–205. Only pasteurized or sterilized milk should be used. All milk products must be protected after preparation from contamination by flies or carriers.

(2) Other foods must be protected from flies and carriers during and after preparation. Raw vegetables and unpeelable fruits should not be served in the Tropics or sub-Tropics where human excreta has possibly been used for fertilizer or where these foodstuffs have been freshened with ditch water. Oysters, unless their source is known to be safe, must not be served raw. Refrigeration at 40° F. or lower should be provided for all food in which bacteria can multiply.

(3) Fly breeding should be energetically combatted at all times. All kitchens and mess halls should be properly screened, with screen doors opening outward, and all crevices closed. Fly traps, flypaper, sprays, and flyswatters should be used in kitchens and mess halls by specially assigned personnel. All foods should be covered whenever feasible.

(4) Sanitary disposal of excreta must be carried out strictly both in fixed installations and in the field. (See AR 40–205.)

(5) Permanent food handlers should be examined in accordance with AR 40–205 before employment, and at regular intervals thereafter. All food handlers must be carefully instructed and trained in personal cleanliness, especially the proper cleansing of hands after defecation.

(6) Cases must be treated under careful enteric isolation precautions with sterilization of excreta and bed linen, protection from flies, and personal hygiene of attendants. Convalescents must not be discharged until stool and urine cultures are negative. They should be recalled for periodic cultures in order to detect possible carrier state. (See AR 40–205 and 40–210.)

(7) Sources of infection should be determined by epidemiological investigation wherever possible, with particular emphasis on the discovery of carriers.

(8) Vaccination with triple typhoid vaccine must be carried out according to AR 40–210. (See sec. VII.)

71.19. Paratyphoid fevers.—*a. Etiologic agent.*—The paratyphoid organisms are *Bact. paratyphosum A* (*Salmonella paratyphi*), *Bact. paratyphosum B* (*Salmonella schottmuelleri*), and other organisms of the genus *Salmonella.*

b. Geographical distribution.—World-wide.

c. Transmission.—Same as for typhoid fever.

d. Recognition.—Same as for typhoid fever The paratyphoid fevers are usually milder than typhoid, with fewer complications and a shorter course. Bacteremia frequently does not occur. There are no clinical differentiating features between the fevers caused by different varieties of paratyphoid organisms. It must be borne in mind that the *Salmonella* organisms can also cause severe bacterial food poisoning and diarrhea, a clinical picture entirely different from paratyphoid fever and also more prevalent in both civil and military life. (See par. 58.)

e. Specific diagnosis.—Same as for typhoid fever.

f. Treatment.—Same as for typhoid fever.

g. Prevention.—Same as for typhoid fever.

71.20. Yaws.—*a. Etiologic agent.—Treponema pertenue.*

b. Geographical distribution.—Common in the Tropics, especially in Africa, Polynesia, the Philippines, and some parts of the Western Hemisphere. Prevalent in the West Indies (especially in Jamaica, Haiti, Trinidad, Antigua, and other islands of the Leeward group), and in coastal and valley settlements of Colombia.

c. Transmission.—By direct contact with lesions of the patient and by nonbiting flies which convey infective material from the open lesions of the yaws patients.

d. Recognition.—(1) An initial granulomatous lesion (mother yaw) usually appears on exposed part of body.

(2) Generalized or secondary lesions appear in 2 to 8 weeks. These resemble raspberries (frambesiform) or large warts, but smaller papules, or scaly, ringworm-like lesions may appear.

(3) Later, disabling lesions consisting of hyperkeratoses with fissuring or ulceration of the plantar epithelium may develop. Gummatous lesions like those of syphilis may occur late in the course of the disease (gangosa—partial destruction of the nose).

e. Specific diagnosis.—The initial and secondary lesions generally contain *Treponema pertenue*, identified by darkfield examination. The blood Wassermann and Kahn reactions usually become positive 1 or 2 weeks after appearance of the initial lesion.

f. Treatment.—(1) Yaws responds to the same therapy commonly used for early syphilis, but good results are usually obtained with much less treatment than in syphilis.

(2) The preferred arsenical is mapharsen. Adult dose for males is 0.06 gram, and for females 0.04 gram. Neoarsphenamine is also effective, 0.75 gram for adult males and 0.6 gram for adult females. The preferred bismuth preparation is bismuth subsalicylate in oil, dose 0.2 gram. The dose for children should be reduced according to age and weight.

(3) The following standard course of treatment for yaws is recommended: four weekly injections of mapharsen or neoarsphenamine and bismuth subsalicylate given on the same day. This is to be followed without a rest period by four weekly injections of mapharsen or neoarsphenamine alone, which in turn is to be followed by eight weekly injections of bismuth subsalicylate alone. Take serologic test, if possible, with eighth and sixteenth treatment.

(4) Follow the patient by clinical examinations and serologic tests at monthly intervals for 3 months, and then at intervals of 3 months for 1 year. If a clinical relapse occurs or if the serologic test remains positive for 6 months after treatment has been started, repeat course of treatment outlined above.

g. Prevention.—Avoid contact with infective open lesions and take precautions to prevent insect transmission. If troops are stationed in close proximity to heavily infected native populations, special care should be given to prevent the contamination of open sores and wounds by infective material. While uncommon, cases have been reported in the white race.

71.21. Bejel.—*a. Etiologic agent.—Treponema pallidum.*

b. Geographical distribution.—Syria, Iraq, and Arabia.

c. Transmission.—Direct contact; possibly also by flies and other insect vectors, and by indirect contact through unsanitary eating and drinking habits. The exact portal and mechanism of entry has not been determined. Usually acquired in childhood, seldom congenital; nonvenereal and rural.

d. Recognition.—Typical primary lesions such as are observed in yaws and venereal syphilis have not been seen in bejel. Early lesions mucocutaneous; late lesions—skin ulcers, periostitis of long bones, symmetrical depigmentation of skin, hyperkeratoses, juxta-articular nodules, erosions of pharynx and nasal bones; cardiovascular and nervous systems apparently not affected.

e. Specific diagnosis.—Darkfield examination of exudate from mucocutaneous lesions in early and relapsed cases positive for *T. pallidum.* Blood Wassermann and Kahn reactions become positive within a few weeks of the onset, and usually remain so whether late lesions appear, or the disease continues latent.

f. Treatment.—Same as for yaws.

g. Prevention.—Same as for yaws.

71.22. Yellow fever.—*a. Etiologic agent.*—The virus of yellow fever.

b. Geographical distribution.—Yellow fever has previously occurred repeatedly in severe epidemic form in most of the countries of the Western Hemisphere and in Africa. Within recent years it has been shown to be endemic in extensive jungle areas in tropical South America (Bolivia, Brazil, Colombia, Ecuador, Paraguay, Peru, Venezuela, possibly in the Guianas, Eastern Panama, and elsewhere), and in Africa. The transfer of virus from such jungle areas to rural or urban regions where *Aedes aegypti* breed and the inhabitants are susceptible has resulted in epidemics of yellow fever.

c. Transmission.—Within recent years it has been shown that there are important differences between the transmission of endemic (jungle) and that of epidemic (urban) yellow fever.

(1) *Jungle yellow fever.*—Endemic yellow fever is transmitted by other mosquitoes as well as *Aedes aegypti* in tropical or subtropical forest environment. In man there may be scattered cases or extensive epidemics. The disease is maintained chiefly by susceptible forest animals. In Brazil *Aedes scapularis*, *A. fluviatilis*, *A. leucocelaenus*, and *Haemogogus capricornii* have been incriminated as effective vectors, and the last two have been found naturally infected. Other species of *Aedes* and other genera native to North America, Europe, Africa, and the Orient have also been proved to be efficient vectors in the laboratory.

(2) *Urban yellow fever.*—This is the *aegypti*-transmitted classical disease of cities, large towns, and occasionally of rural areas. Where susceptible individuals and *Aedes aegypti* are numerous the urban disease may appear in epidemic form after the disease is introduced from areas of jungle or urban infection. Epidemics may also result from the introduction of nonimmune troops into places in which the endemicity of urban yellow fever is barely perceptible. *Aedes aegypti* are found in the Tropics, sub-Tropics, and temporarily elsewhere, if newly introduced, during a warm season.

d. Recognition.—Incubation period 3 to 6 days; onset sudden; high fever with short remission third to fourth day followed by recrudescence; pulse rate at first rapid but begins to fall with a rising temperature; albuminuria is usually marked on third day; black vomit; skin and mucous membrane hemorrhages; jaundice of increasing intensity. Mortality: 10 to 30 percent.

e. Specific diagnosis.—A provisional diagnosis may be made on the basis of symptoms. For a final decision, 30 cc of blood should be drawn early in the disease and again 3 weeks after onset. The sterile blood specimens should be sent to a laboratory equipped to make yellow fever protection tests. If the first fails to protect mice against yellow fever and the second protects, the disease was yellow fever.

f. Treatment.—(1) *Specific.*—None.

(2) *General.*—(*a*) Isolate the patient in a screened room.

(*b*) In severe cases during the acute state, give only citrus fruit juices and alkaline water (e. g., 3,000 cc tap water with 15 grams bicarbonate of soda daily). In mild and moderate cases a light diet high in carbohydrates and low in fats is recommended.

(*c*) Give saline laxative at onset and thereafter enemas as needed.

(*d*) If vomiting prevents feeding by mouth, give glucose 50 grams in 1,000 cc of physiological salt solution intravenously three times daily, accompanied by thiaminehydrochloride 2 milligrams.

(*e*) When the patient is able to eat he should receive a diet high in carbohydrates and relatively low in fats. Milk, eggs, and cheese are desirable. This diet should be supplemented daily with 4 to 6 multivitamin capsules. This regime should be continued until patient is convalescent. The patient's activities should be resumed gradually and a long period of convalescence allowed.

g. Prevention.—(1) *Immunization.*—Vaccination confers an immunity of several years' duration. All military personnel traveling to, or through, or stationed in areas in which yellow fever is endemic should be vaccinated against the disease. (See sec. VII.)

(2) *Precautions against the transfer of yellow fever from endemic to nonendemic areas.*—Special care must be exercised to prevent the introduction of yellow fever into nonendemic areas through the transfer of infected mosquitoes and human beings. Airplane transportation should be safeguarded by the vaccination of flying personnel, the elimination of mosquitoes in airplanes by suitable spray-killing, and the examination and observation of unvaccinated passengers for signs of infection. See current AAF Regulations for details.

(3) *General measures to be used in face of epidemic.*—(*a*) Isolate patient in room protected with 18-mesh screen for 4 days from onset of symptoms (period of communicability).

(*b*) Destroy all mosquitoes in the quarters in which infection may have occurred, using an approved pyrethrum spray.

(*c*) Isolate persons who have probably been exposed, in quarters protected with 18-mesh screen for 10 days (maximum period of incubation), and inspect daily for onset of suspicious symptoms.

(*d*) Vaccinate immediately against yellow fever all persons in the *A. aegypti* infected area who have not already received vaccine. This is a very important control measure.

(*e*) Arrange for daily inspection of entire military and civilian population in exposed area in order to discover any early cases of yellow fever, remembering that jaundice is seldom an early symptom but that headache and fever are the commonest manifestations at onset.

(*f*) Perform autopsy or viscerotomy on all cases dying of fever of 10 days' duration or less, both in military and surrounding civilian population, and examine tissue from liver for histological evidence of yellow fever.

(*g*) Institute vigorous, organized campaign for the complete suppression of *A. aegypti* breeding. *Aedes aegypti* is a domestic mosquito. It breeds mostly in clear water, in artificial containers such as rain barrels, roof gutters, tin cans, tire casings, flower vases, etc. The eggs are resistant to drying. The adults are found most frequently in human habitations. Its flight range is limited to a few hundred yards. Screen wire of United States standard 18-mesh is required to exclude it.

(*h*) To suppress *aegypti* breeding, the entire camp or community should be divided into districts so that every room of each building and all surrounding premises can be systematically inspected once a week by a trained district inspector. Inspectors should carry flashlight. Fuel oil should be applied to all containers found to harbor *aegypti* larvae, if they are not immediately eliminated

or made permanently mosquitoproof. This procedure makes it necessary to scrub the container and remove the adhering eggs before refilling. Thorough check inspections by reliable supervisor are essential. Such measures are highly effective in quickly rendering the community noninfectible. (See AR 40–205 and 40–210.)

71.23. Diet in treatment of tropical diseases.—In tropical diseases the same dietary principles apply as in all diseases. If the nature of the patient's condition prevents his consuming a wisely constructed diet, deficiencies should be made good with specific supplementary substances administered in such a manner that they will be absorbed.

a. Acute infectious diseases interfere with the intake of food and increase metabolic activities. The requirement for nutritional elements may thereby be increased. This applies to calories, proteins, minerals, and vitamins. Various members of the vitamin B complex are involved in metabolism. They especially enter into the metabolism of carbohydrates. Deficiency disease due to an inadequate supply of factors of the vitamin B complex may be precipitated by fever, vomiting, diarrhea, or the administration of glucose. Therefore, when parenteral injections of glucose are administered, it is desirable to guard against vitamin deficiencies. Each 50 grams of glucose should be accompanied by 2 milligrams thiamine hydrochloride, 50 milligrams ascorbic acid, and 15 milligrams nicotinic acid amide, given separately by mouth or parenterally or in the glucose solution.

b. In chronic disease, when a well balanced diet cannot be administered, supplementary vitamins should be given orally or parenterally. The following daily doses are considered minimal: 2 milligrams thiamine hydrochloride; 3 milligrams riboflavin; 20 milligrams nicotinic acid amide; 75 milligrams ascorbic acid; 5,000 international units vitamin A, and 400 international units vitamin D.

c. The medical officer should watch for early clinical signs of manifest nutritional disease, especially glossitis or other evidence of early pellagra, the ocular symptoms and cheilosis of riboflavin deficiency, and the peripheral neuritis of thiamine deficiency.

(1) When pellagra can be recognized, give 50 milligrams nicotinic acid amide orally each hour for 10 hours. Repeat the following day until the active symptoms disappear. For clinical riboflavin deficiency, give 3 milligrams riboflavin three times daily by mouth. For the peripheral neuritis of thiamine deficiency, give at least 10 milligrams thiamine hydrochloride orally each day for 1 week.

(2) Scurvy, if detected, should be treated by the daily oral administration of 200 milligrams ascorbic acid and the free use of fresh vegetables and citrus fruits.

(3) Vitamin K deficiency, manifested by hemorrhagic phenomena in obstructive jaundice, in severe liver damage of any kind, and in purpura neonatorum, or resulting from severe diarrhea or lack of intestinal absorption and poor secretion of bile salts may be treated with vitamin K (2 milligrams intramuscularly daily) combined with bile salts.

(4) Protein deficiency is encountered in nephrosis, chronic infectious diseases, poor intestinal absorption, severe diarrhea, and in diets poor in protein. If manifested clinically by edema, or acute loss of blood protein, it is best treated by the intravenous administration of human serum or plasma.

(5) Vitamin A deficiency is manifested by night blindness. Treatment consists of the daily administration by mouth of 20,000 units of vitamin A for 4 weeks.

[A. G. 062.11 (3–23–43).] (C 1, May 6, 1943.)

BY ORDER OF THE SECRETARY OF WAR:

G. C. MARSHALL,
Chief of Staff.

OFFICIAL:
J. A. ULIO,
Major General,
The Adjutant General.

U. S. GOVERNMENT PRINTING OFFICE: 194

TECHNICAL MANUAL

GUIDES TO THERAPY FOR MEDICAL OFFICERS

CHANGES No. 2

WAR DEPARTMENT,
WASHINGTON 25, D. C., 31 August 1943.

TM 8–210, 20 March 1942, is changed as follows:

30. Heat exaustion, heat stroke, and heat cramps.—

* * * * * * *

c. (Superseded.) Heat stroke demands prompt treatment. The single most important objective is the lowering of the body temperature as rapidly as possible. All necessary and available measures for the accomplishment of this objective should begin in the field as soon as the the condition is recognized. They should be continued without interruption during transportation of the patient to and after his arrival at the hospital until the rectal temperature has been lowered to (but not below) 102° F. In the treatment of the patient, the entire body should be sprayed or sprinkled with water, rubbed briskly to increase circulation to the skin, and fanned continually to increase the speed of evaporation and the consequent loss of heat. Ice or ice water should not be employed in any form in this connection. When the temperature has been reduced from the critical levels, the patient must be closely observed for several days to guard against relapses.

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[A. G. 300.7 (2 Aug 43).] (C 1, 31 Aug 43.)

BY ORDER OF THE SECRETARY OF WAR:

G. C. MARSHALL,
Chief of Staff.

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U. S. GOVERNMENT PRINTING OFFICE: 1943

TM 8–210

TECHNICAL MANUAL

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GUIDES TO THERAPY FOR MEDICAL OFFICERS

UNITED STATES
GOVERNMENT PRINTING OFFICE
WASHINGTON : 1942

For sale by the Superintendent of Documents, Washington, D. C.

FOREWORD

The Medical Department of the United States Army has availed itself of the advisory and consultative facilities of the National Research Council of the National Academy of Sciences in the conduct of professional care of the Army during the present national emergency. The material on which the text of this manual is based was furnished largely by the various committees and subcommittees of the Division of Medical Sciences, National Research Council, with the assistance of other consultants.

In 1863 President Lincoln established the National Academy of Sciences in order to have the advice of the leading scientists of the country in problems of national concern. In 1916 the Academy offered its services for national defense and at the request of President Wilson it created the National Research Council as its active agent. In 1918 President Wilson by executive order requested the National Academy of Sciences to perpetuate the National Research Council.

Early in 1940 The Surgeons General of the Army and Navy requested that the Division of Medical Sciences of the National Research Council establish committees which should act in an advisory capacity to the two medical corps. The material compiled by many of these committees is incorporated in this manual and was edited by Dr. R. N. Nye of the Committee on Information. Final compilation was conducted by the pertinent divisions of the Office of The Surgeon General, United States Army.

In section II, Surgical Emergencies, the following subcommittees of the Committee on Surgery and consultants were contributors for these various subjects: "Flesh Wounds" and "Burns," the Subcommittee on Surgical Infections, Drs. Frank L. Meleney, Chairman, W. M. Firor, Sumner Koch, J. S. Lockwood, Perrin H. Long, Champ Lyons, and Roy H. McClure, and also Dr. W. F. Rienhoff, Jr.; "Wounds of the Head and of the Nervous System," the following members of the Subcommittee on Neurosurgery, Drs. Howard C. Naffziger, Chairman, Charles Bagley, Jr., Gilbert Horrax, Cobb Pilcher, Tracy J. Putnam, and Byron P. Stookey, and also Dr. Foster Kennedy, Chairman

of the Subcommittee on Neurology; "Wounds and Injuries of the Eye," the Subcommittee on Ophthalmology, Drs. Harry S. Gradle, Chairman, W. L. Benedict, S. R. Gifford, P. C. Kronfeld, and L. T. Post; "Wounds and Injuries of Ear, Nose, and Throat," the Subcommittee on Otolaryngology, Drs. Harris P. Mosher, Chairman, G. M. Coates, R. A. Fenton, W. E. Grove, F. T. Hill, C. J. Imperatori, V. H. Kazanjian, S. J. Kopetzky, H. I. Lillie, J. J. Shea, B. R. Shurley, and W. P. Wherry; "Maxillofacial Wounds," the Subcommittee on Plastic and Maxillofacial Surgery, Drs. Robert H. Ivy, Chairman, J. Staige Davis, J. D. Eby, P. C. Lowery, and Ferris Smith; "Wounds of the Chest," the Subcommittee on Thoracic Surgery, Drs. Evarts A. Graham, Chairman, Isaac A. Bigger, E. D. Churchill, and Leo Eloesser; "Abdominal Wounds," Dr. Alton Ochsner of the Committee on Surgery; "Wounds of the Genito-urinary System," the Subcommittee on Urology, Drs. Herman L. Kretschmer, Chairman, William Braasch, H. G. Hamer, Frank Hinman, O. S. Lowsley, and A. J. Scholl; "Wounds of the Large Blood Vessels," the Subcommittee on Vascular Injuries, Drs. John Homans, Chairman, A. W. Allen, Geza de Takats, D. C. Elkin, and W. G. Maddock; "Sprains, Strains, and Contusions," Dr. Augustus Thorndike, Jr., of Boston; "Fractures," the Subcommittee on Orthopedic Surgery, Drs. G. E. Bennett, Chairman, LeRoy C. Abbot, Harold Conn, William Darrach, R. H. Kennedy, John A. Key, F. C. Kidner, Guy Leadbetter, P. B. Magnuson, M. N. Smith-Petersen, and P. D. Wilson; "Relief of Pain" and "Respiratory Emergencies," the Subcommittee on Anesthesia, Drs. Ralph M. Waters, Chairman, L. S. Booth, John S. Lundy, E. A. Rovenstine, and R. M. Tovell, and also Dr. Charles Carrol Lund of Boston; "Secondary or Wound Shock," Dr. Alfred Blalock, Chairman of the Subcommittee on Shock.

Section III, Medical Emergencies, was prepared by the Subcommittee on Diagnosis and Therapeutics of the Committee on Medicine, Drs. Warfield T. Longcope, Chairman, Hugh Morgan, M. C. Pincoffs, and W. B. Porter, Dr. O. H. Perry Pepper, Chairman of the Committee on Medicine, was at that time Chairman of the Subcommittee on Diagnosis and Therapeutics, and the section was prepared under his direction. The paragraph on "Acute Psychoses and Related Conditions" was prepared by Dr. Edward A. Strecker of the Subcommittee on Psychiatry.

Section IV, Diagnosis and Treatment of Venereal Diseases, was prepared by the Subcommittee on Venereal Diseases, Drs. J. E. Moore, Chairman, E. P. Alyea, C. W. Clarke, O. F. Cox, Jr., J. F. Mahoney, U. S. Public Health Service, Nels Nelson, and J H. Stokes.

Section V, Chemotherapy and Serotherapy in Certain Infectious Diseases, was prepared by the Committee on Chemotherapeutic and Other Agents, Drs. Perrin H. Long, Chairman, Francis Blake, J. S. Lockwood, J. F. Mahoney, U. S. Public Health Service, and E. K. Marshall, Jr.

Section VI, Treatment and Control of Certain Tropical Diseases, was prepared by the Subcommittee on Tropical Diseases, Drs. Henry E. Meleney, Chairman, Mark Boyd, E. H. Hume, Thomas T. Mackie, W. A. Sawyer, and R. B. Watson.

Section VII, Rickettsial Diseases, was prepared by Dr. Rolla E. Dyer, U. S. Public Health Service, of the Subcommittee on Infectious Diseases.

TECHNICAL MANUAL
No. 8-210

WAR DEPARTMENT,
WASHINGTON, March 20, 1942.

GUIDES TO THERAPY FOR MEDICAL OFFICERS

SECTION I

GENERAL

1. **Purpose and scope.**—The purpose of this manual is to provide the medical officer with a handy text containing guides to therapy under emergency conditions or in diseases with which he is relatively unfamiliar. Much of the text is based on recommendations made, at the request of The Surgeon General, by various committees of the Division of Medical Sciences, National Research Council; in fact three of the sections are somewhat modified versions of circular letters already released by the Office of The Surgeon General. The accepted

treatment of the diseases commonly encountered in civilian life is not discussed, nor are such topics as the control of communicable diseases and sanitation, since they are adequately covered, respectively, by Reprint No. 1697, "Public Health Reports," and FM 8–40, to which the medical officer is referred. Furthermore, it should be emphasized that these guides to therapy are not rules or regulations that must be followed but merely suggestions to be used at the discretion of the medical officer.

SECTION II

SURGICAL EMERGENCIES

2. General.—The following paragraphs in this section contain guides to the treatment of wounds of various parts of the body, to the care of other types of injury, and to certain types of general therapy applied to diseased conditions that are usually associated with wounds and injuries. These guides cover first-aid and emergency treatment that can and should be administered at battalion-aid, collecting, and clearing stations and surgical hospitals. Nontraumatic surgical emergencies are not considered. It is obvious that no definite rules can be suggested for a given case; however, these general guides should be helpful to the medical officer.

3. Flesh wounds.—*a. General.*—(1) The term "flesh wounds" includes wounds of the skin, subcutaneous tissue, muscles and tendons, nerves (par. 4), and blood vessels (par. 11). It does not

concern wounds of body cavities, such as the pleural cavities and the abdomen, or fractures of bones, which are dealt with elsewhere (pars. 8, 9, and 13).

(2) Flesh wounds may be incisions, punctures, lacerations, or contusions. They may be caused by a sharp object, such as a knife or bayonet, by a blunt object, such as a shell fragment, or by a torsion of the tissues from a heavy blow. They may be extensive and superficial, or the wound entrance may be small, with extensive damage to the deep tissues. If blood vessels are punctured or torn by an object entering through a small opening, there may be extensive extravasation of blood through the tissues.

(3) Various kinds of bacteria that were on the skin surface may be carried in when the wound is made, as well as organisms originally on the object causing the wound. Bits of clothing highly contaminated with soil or intestinal organisms are often present and bacteria may get in after the original injury, if the wound is not properly protected by dressings.

b. Principles of treatment.—(1) Hemorrhage must be controlled.

(2) Contaminating bacteria must be removed as completely as possible.

(3) Injured tissue must be débrided, since it harbors organisms, is a favorable medium for their growth, and is slow to heal.

(4) Further injury and further contamination with bacteria must be avoided.

c. Treatment.—(1) Relieve pain (par. 14).

(2) Except for minor wounds, anesthetize the patient to remove properly the damaged tissue.

(3) Control hemorrhage. A tourniquet should be applied above the wound if hemorrhage cannot be adequately controlled by the application of a firm dressing; it should be released every hour for a few minutes, provided the bleeding can be controlled by digital pressure during release; if this is impossible the ruptured vessel should be litigated as soon as possible (par. 11).

(4) If not previously applied, protect the wound from further contamination with a sterile gauze compress.

(5) Administer the necessary prophylactic measures against pyogenic, tetanus, and gas-bacillus infections (pars. 48, 49, 50, and 51).

(6) If necessary, treat for shock (par. 15).

(7) As soon as possible, wash the skin thoroughly with neutral soap and water, remove grease or dirt with benzene, and rub the skin with sponges wet with 70 percent alcohol until the sponges come away clean.

(8) Débride the wound under strict aseptic precautions, the injured skin margins being trimmed back and all the damaged tissue on the wound surface, including any tissue densely infiltrated with blood, being removed, as well as all blood clots and foreign bodies, so that nothing but grossly normal tissue is left on the wound surface; a clean set of instruments should be used for the dissection of the deep portions of the wound. (All the débrided tissue and foreign bodies should be sent, whenever practicable, to the bacteriologic laboratory for study, so that the surgeon may be informed concerning the contaminating bacteria as soon as possible.)

(9) Clamp all bleeding vessels, and tie them with fine catgut or silk sutures.

(10) After débridement, *not* before, forcefully wash out the wound with large amounts of sterile physiologic saline solution, to remove all the loose particles of tissue and blood.

(11) Dust the wound with sulfanilamide powder (par. 51).

(12) Pack the wound lightly with sterile gauze and cover with an encircling dressing of sterile sheetwadding sufficiently wide to prevent exposure of the wound during transit; in injuries of the extremities, the tips of the fingers or toes should be left exposed for inspection as to circulatory adequacy.

(13) When necessary, use plaster or other splints, if available, to immobilize the part; this applies particularly when muscles and tendons have been cut. A pressure dressing should be applied where indicated.

(14) Under combat conditions, no attempt should be made to close any part of a wound or to suture cut nerves or tendons. If time is not a factor, the ends of cut nerves and tendons should be approximated wherever indicated, thereby improving the chances of a favorable outcome. (par. 4).

4. Wounds of head and nervous system.—*a. General.*—(1) Time is of the utmost importance in the treatment of cases with severe injuries of the head or of the nervous system. Certain types of emergency treatment can and should be given, but these procedures should be done as thoroughly and as quickly as

possible so that the patient can be transferred rapidly, with little or no disturbance during transportation, to a hospital where proper care is available.

(2) The three primary purposes of emergency treatment are to—

(*a*) Combat conditions, such as hemorrhage and shock, that constitute an immediate danger to life.

(*b*) Prevent infection.

(*c*) Transport the patient with all possible speed to a hospital where complete care can be given and where he can be kept under close observation.

(3) Secondary considerations are to—

(*a*) Relieve pain.

(*b*) Attempt to prevent infection by the administration of specific prophylactic agents.

(*c*) Prevent additional injury by immobilization, by the placing of an inlying catheter, if indicated, and by protection against decubitus ulcers.

(*d*) Perform primary closure of superficial wounds, if time and facilities permit.

b. Treatment.—(1) *General.*—(*a*) Keep the patient warm by whatever means available.

(*b*) Lay the unconscious patient on his side in such a position that the breathing is free; no patient should be fed by mouth until it has been shown, by having him suck a wet swab, that his swallowing mechanism is unimpaired.

(*c*) Relieve pain (par. 14). Morphine or opium in any form should *never* be given in cases of head injury; it may be given freely in cases with spinal injury below the cervical region.

(*d*) For restlessness, up to 0.3 gram (5 grains) of phenobarbital sodium parenterally may be given, but for delirium, 30 cc (1 ounce) of paraldehyde or 2 grams (30 grains) of chloral hydrate by rectum may be necessary; convulsions, which are infrequent shortly after injury, can be treated with phenobarbital sodium or a few whiffs of chloroform.

(*e*) Remove all field dressings, since they are apt to be contaminated and are often applied too tightly.

(*f*) Superficial wounds may be cleansed with soap and water, but wounds with any possible connection with the meningeal spaces should be undisturbed except for fresh sterile dressings.

(*g*) The treatment of shock (par. 15), the administration of sulfanilamide and its derivatives and of specific prophylactic agents for tetanus and gas-bacillus infection (par. 51), and similar general measures should follow the course employed for other wounds, *with the exception of sedative and analgesic drugs* (see above).

(*h*) Make a brief notation on the patient's record of gross neurologic findings (state of consciousness, pulse rate, size of pupils, paralysis, and other neurological signs), the character of the wound, and *the amount and time of administration of any medication.*

(2) *Superficial wounds.*—This category includes wounds of the head or back in which there is no evidence of fracture or of perforation of the meningeal spaces.

(*a*) If such wounds are seen early and are relatively clean, and if time and facilities permit, they should be thoroughly cleansed and débrided, and closed, without drainage, with interrupted silk sutures.

(*b*) If closure is impracticable, the hair should be clipped (or better, shaved), the wound cleansed, with the removal of foreign bodies, dirt, and hair, and a vaseline-gauze dressing applied.

(*c*) Control hemorrhage by a tight bandage.

(*d*) If these measures cannot be carried out under novocain anesthesia, the wound should be inspected, cleaned superficially, a clean vaseline-gauze dressing applied, and the patient evacuated as rapidly as possible to a hospital where complete care can be given.

(*e*) It should *always* be remembered that every scalp wound, no matter how trifling, is a potential penetrating wound of the skull and that many penetrating wounds are found among those of the wounded who are walking.

(3) *Severe head wounds.*—(*a*) Patients with wounds in which the skull bones are extensively damaged or the dura penetrated should be evacuated to a hospital with all possible speed.

(*b*) The most important first-aid measure is to avoid all disturbance and handling of the wound until the operator is prepared to follow his cleansing measures with immediate and adequate débridement; this is usually possible only in completely equipped hospitals.

(*c*) Patients with this type of wound should be subjected to a minimum of manipulation and should be handled as gently

as possible; unless the patient is in shock, slight elevation of the head is advantageous.

(4) *Severe spinal wounds.*—(*a*) Of primary importance in this type of wound is the handling of the patient. The slightest displacement of fractured vertebral fragments by manipulation may result in irreparable damage to the spinal cord. Litter bearers and orderlies should be cautioned, and movement of the patient should be limited to an irreducible minimum. Potential damage to the cord by fractured fragments or foreign bodies may exist where no paralysis or anesthesia is present at first. For this reason, even apparently minor injuries of the back should be handled with great care until absence of fracture or cord injury is established.

(*b*) The patient should be placed on the litter in such a way that moderate extension of the spine is obtained; this may be accomplished with the patient prone or recumbent, but in the latter position a rolled blanket or pillows *must* be placed under the site of injury. If an obvious kyphos exists, no pressure should be made over it. Lifting of the patient to and from the litter should be rigorously avoided; *the patient should be rolled on.* A support should be placed under the forehead, and sandbags should be placed on each side of the head and neck to prevent movement, particularly if the cervical spine is involved. Do not turn the patient's head sideways.

(*c*) Except for the most superficial wounds without evidence of cord injury, no débridement and suture should be attempted; such cases should be redressed and immediately evacuated to a properly equipped hospital.

(*d*) Superficial wounds should be cleansed with soap and water, and alcohol.

(*e*) On no account should the spinal cord be touched, if it is exposed.

(*f*) If packing is necessary to control hemorrhage, pressure must not be applied to the cord itself; even severe hemorrhage is less dangerous.

(*g*) If cutaneous anesthesia is present, the skin *must* be kept dry and all bony prominences should be protected from pressure, so far as possible, by rubber rings or gauze "doughnuts," to prevent decubitus ulcers.

(*h*) If paralysis of the bladder is present, the bladder should *never* be emptied by manual pressure above the symphysis; a

catheter should be inserted, and tidal drainage should be instituted as soon as possible. The time of placing the catheter should be noted on the patient's record.

(*i*) In the presence of cord injury, early operation may prevent a fatality or permanent paraplegia; hence, all possible speed should be employed in evacuating such cases to the rear.

(5) *Wounds of peripheral nerves.*—(*a*) Wounds of the extremities that are likely to have divided peripheral nerves are essentially of two varieties: through-and-through wounds from shell fragments or bullets, and widely open, lacerated wounds.

(*b*) The former should have merely the usual cleansing and a fresh sterile dressing.

(*c*) The latter should be irrigated freely with sterile physiologic saline solution to get rid of gross particles of dirt and foreign substances (occasionally dirty tags of skin may be cut off). The wound should then be covered with a vaseline-gauze dressing, and the patient should be evacuated as rapidly as possible to a hospital.

(*d*) In rare cases, a partly or completely divided nerve may lie exposed in the wound. If so, and if time permits, suturing should be carried out at once with mattress stitches of the finest silk available. The stitches should include only the nerve sheath. One must always be sure not to unite a divided nerve with a divided tendon.

(*e*) Mobilization of nerves and extensive dissection to permit end-to-end suture is never to be done as part of primary wound débridement; these operations are a part of the final treatment after patients have been sent to hospitals.

(*f*) The foregoing policies may be altered by the coexistence of extensive fractures or injuries of large arteries; in such cases, the nerve injury may be regarded as of secondary urgency.

(*g*) Since most large arteries are closely associated with one or more major nerve trunks, in clamping them *great care must be taken to avoid injury of the accompanying nerve or nerves.*

(*h*) In the presence of peripheral nerve injury, the involved extremity should be placed in such a position that minimum retraction of the divided nerve ends takes place, and the extremity should then be immobilized.

5. Wounds and injuries of eye.—*a. Lacerations of eyelids and surrounding soft tissues.*—(1) Gently cleanse with irri

gations of any sterile nonirritating solutions (physiologic saline solution or 4 percent boric acid).

(2) Approximate the edges of the wound and hold them in place with a firm bandage.

(3) Do *not* sew the edges of the wound together.

(4) Proper prophylactic measures to avoid tetanus should be instituted in wounds about the eye, though they may not be extensive in character (par. 49).

b. Foreign bodies on surface of eyeball.—(1) Anesthetize the eye with a 1 percent solution of butyn or a 0.5 percent solution of pontocaine.

(2) Wipe the foreign body off *gently* with a cotton-wound applicator, moistened with a mild antiseptic solution (4 percent boric acid).

(3) If this is impossible, try to remove the foreign body with a sterilized eye spud, if available.

(4) Instill a mild antiseptic solution (4 percent boric acid).

(5) Do *not* try to remove a foreign body without proper light and magnification.

(6) *Remember* that a foreign body on the cornea is often followed by a serious sight-destroying ulcer if neglected or if removed under nonsterile conditions.

c. Perforating wounds of eyeball.—(1) Anesthetize the eye, as above.

(2) Remove any superficial foreign bodies, as above.

(3) Flush the eye with a mild antiseptic solution (4 percent boric acid).

(4) Close the eyelids and apply a fairly firm dressing, held in place by adhesive tape.

(5) *Evacuate as rapidly as possible*, so that the patient may receive proper ophthalmologic care promptly.

(6) Do *not* close the wound with sutures.

(7) Do *not* excise any tissue protruding from the wound.

(8) Do *not* cover the wound with a conjunctival flap, unless specifically equipped.

d. Contusions and nonperforating injuries of eyeball.—(1) Manipulate as little as possible.

(2) Try to determine whether there is a perforation.

(3) Evacuate the patient to proper ophthalmologic care as promptly as possible.

(4) If such injuries are made light of or neglected, they frequently lead to complete loss of sight.

e. Gas injuries of eyes.—(1) Flush out the eyes with *large amounts* of a clean, nonirritating solution (physiologic saline solution or 4 percent boric acid) *as soon as possible.*

(2) Fill the eye with any nonirritating sterile grease (vaseline).

6. Wounds and injuries of ear, nose, and throat.—*a. Ear.*—(1) *Contusion of auricle.*—Blows on the auricle often cause a hematoma between the skin and attached soft tissues and the cartilage. If the hematoma becomes infected there is a progressive destruction of the cartilage, which results in a shriveled ear.

(*a*) Evacuate the hematoma early by syringe or incision.

(*b*) Apply a pressure bandage. There should be an ample bed of gauze behind the auricle to help preserve its form.

(2) *Cuts and lacerations of auricle.*—(*a*) Clean according to surgical principles (see par. 3).

(*b*) Suture lightly to allow drainage, taking care that the suture does not pass through the cartilage.

(*c*) Embed the auricle in gauze, and apply a bandage.

(3) *Insects in external auditory canal.*—(*a*) Fill the canal with oil to drown the insect, or plug the canal externally with cotton saturated with a few drops of ether or chloroform.

(*b*) Syringe.

(4) *Furunculosis of canal.*—Furunculosis of the canal may appear suddenly and, in the beginning may suggest mastoiditis. The patient complains that the ear "feels full," and there is pain in and about the ear, with swelling behind and in front of the auricle; however, the swelling behind the ear is not markedly tender as it is in mastoiditis. Movement of the auricle elicits pain. The canal is reddened, tender, and symmetrically narrowed until the infection localizes and the boil comes to a head. Boils often come in crops.

(*a*) Do *not* incise the canal.

(*b*) Irrigate the canal with hydrogen peroxide and pack lightly with gauze smeared with 10 percent ichthyol ointment.

(*c*) If feasible, apply heat.

(5) *Foreign bodies in external auditory canal.*—(*a*) Soft, foreign bodies that are not impacted are removed by syringing, small hooks, or appropriate forceps.

(*b*) Impacted foreign bodies, like pieces of wood, stone, or metal, should always be removed by exposing them by an incision through the posterior wall of the canal. The incision is made opposite the canal in the auriculomastoid groove and carried forward through the canal in front of the foreign body.

(6) *Rupture of tympanic membrane.*—(*a*) Clean the auricle only.

(*b*) Do *not* syringe the canal.

(*c*) Plug the canal superficially with sterile cotton or gauze.

(7) *Bleeding from external auditory canal.*—Bleeding from the external auditory canal not caused by a laceration of the soft structures of the canal or injury by a foreign body is due to a fracture of the temporal bone and occurs in severe head injuries (par. 4).

(*a*) Clean the auricle only.

(*b*) Do *not* syringe the canal.

(*c*) Plug the canal superficially with sterile cotton or gauze.

(*d*) Evacuate the patient to a hospital.

(*e*) If the facial nerve is injured by the fracture of the skull, no immediate specific treatment is indicated.

(8) *Massive wounds of mastoid process.*—Deep cleaning of an extensive mastoid wound should be left to an otologist, since in this procedure both the facial nerve and the lateral sinus are involved.

(*a*) Clean the wound superficially, according to surgical principles (par. 3); strictly *avoid* the use of cleansing solutions.

(*b*) Leave the wound open, pack with sterile gauze, and apply a bandage.

(*c*) Evacuate the patient to a hospital.

(9) *Sudden deafness.*—(*a*) Sudden deafness, unilateral or bilateral, partial or complete, may be due to cerumen sealing one or both canals. If so, remove the cerumen by syringing the canal with warm physiologic saline solution. If the cerumen is hard and tightly impacted, soften it before syringing by filling the canal with hydrogen peroxide, allowing this to remain in the canal a few minutes.

(*b*) Sudden deafness, unilateral or bilateral, partial or complete, after exposure to excessive noise is due to injury of the internal ear. Hemorrhage into the closed systems of the internal ear is the usual cause of the injury. The deafness is

accompanied by nystagmus, tinnitus, and vertigo. In such a case, evacuate the patient to a hospital for care by an otologist.

b. Nose.—(1) *Fracture.*—(*a*) Simple fractures of the nose with the characteristic lateral deformity and some depression of the bridge are easily reduced when treated early.

(*b*) Introduce a flat spatula-like instrument into the nose on the side of the lateral deformity, and make upward pressure on the under surface of the nasal bone, raising it until the overlapping of the nasal bone and the edge of the frontal process of the superior maxilla is reduced. When this happens the nasal bones can be easily forced back into the midline, and will almost always stay in place without the support of a nasal packing.

(2) *Severe flattening.*—No immediate treatment is indicated; evacuate the patient to a hospital, for treatment by a maxillo-facial surgeon.

(3) *Severe compound fractures.*—(*a*) Control hemorrhage.

(*b*) Apply a protective dressing.

(*c*) Evacuate the patient to a hospital.

(4) *Penetrating wounds of accessory sinuses.*—(*a*) Cleanse the wound according to surgical principles (par. 3), avoiding the use of cleansing solution so far as possible, especially in penetrating wounds of the frontal sinus.

(*b*) Do *not* syringe the frontal sinus cavity.

(*c*) Pack each sinus to stop bleeding.

(*d*) Leave the wound open, and apply a sterile-gauze dressing and a bandage.

(*e*) Evacuate the patient to a hospital.

(5) *Fracture of cribriform plate.*—After severe trauma to the skull, with or without an external wound, an intermittent or continuous watery drip from one or both sides of the nose indicates fracture of the cribriform plate.

(*a*) Do *not* syringe the nose.

(*b*) Protect the nasal cavity by an external sterile pad, changing the pad frequently, if possible.

(*c*) Evacuate the patient to a hospital.

c. Throat.—(1) *Fracture of hyoid bone.*—(*a*) If there is much dyspnoea, perform a tracheotomy.

(*b*) If there is much difficulty in swallowing, feed the patient through a nasal or pharyngeal tube.

(*c*) Evacuate the patient to a hospital.

(2) *Fracture of larynx.*—(*a*) If there is much continuous dyspnoea, perform a tracheotomy.

(*b*) Evacuate the patient to a hospital.

(3) *Open and destructive wounds of larynx.*—(*a*) Treat the wound according to surgical procedure (par. 3).

(*b*) If there is increasing dyspnoea or marked dyspnoea, perform a tracheotomy.

(*c*) Evacuate the patient to a hospital.

(4) *Window tracheotomy.*—Time permitting, a window tracheotomy, without the insertion of a tracheotomy tube, greatly simplifies the aftercare of any patient on whom tracheotomy is performed; it is especially useful in destructive wounds of the larynx or trachea. If the stumps of the isthmus of the thyroid gland are properly ligated, so that there is no bleeding into the trachea, the surgeon is spared the first-night worries and most of the subsequent worries that accompany the customary tracheotomy, with the insertion of a tube. The steps of the window tracheotomy are as follows:

(*a*) Expose and mobilize the isthmus of the thyroid for at least 1 inch.

(*b*) Clamp the isthmus on both sides of the midline, and cut it in the midline between the clamps.

(*c*) Suture each stump of the isthmus with interrupted sutures, making sure that all bleeding is controlled; if the latter is not accomplished, the whole purpose of the operation is defeated.

(*d*) Turn the stumps of the isthmus outward, and suture them to the pretracheal muscles and to the skin; this should leave the front of the trachea widely exposed and dry.

(*e*) Make a crucial incision in the trachea, and remove the four triangular flaps of the tracheal wall with a knife or punch, leaving a tracheal opening at least 1¼ inches in diameter.

(*f*) The opening remains sufficient for about a week; by then the pretracheal muscles have returned to the midline, and the insertion of a tracheotomy tube is required if the tracheal opening is still necessary.

7. Maxillofacial wounds.—*a. General.*—(1) A correlated plan of treatment, if carried out from the time the wound is incurred until definitive treatment is available, will greatly shorten the period of disability of patients with jaw injuries, and a larger number will be restored to approximately normal

function and appearance than if haphazard methods are followed. Certain things should be done and others should not be done; hence, attention to these points will save many lives and facilitate later treatment by specialists.

(2) Every officer and soldier of the Medical Department in the combat zone is supplied with equipment useful in rendering first-aid treatment for jaw injuries. The first-aid packet adapts itself admirably for jaw fractures and can be used for a great many types of gunshot wounds of the face and head. The compress, sewn to the central portion, can be made to serve as a hammock or sling to support the injured structures. By tearing the attached bandage lengthwise, the dressing becomes an ideal 4-tailed bandage, which may be securely and satisfactorily applied by an officer or enlisted man. The compress itself can be separated from the bandage and used as an extra packing, dressing, or support over any region. The safety pins assist in making the dressings secure. With the aid of common rubber bands or elastics and the safety pins, emergency fixation can be applied with bandages or adhesive tape.

b. First-aid treatment.—The points demanding special attention in first-aid treatment may be formulated as follows:

(1) Arrest of hemorrhage.

(2) Provision of adequate respiratory airway.

(3) Temporary approximate reduction and fixation of bone fragments.

(4) Provision of safe transportation to the hospital.

(5) Relief of pain, treatment of shock, and other general emergency measures are applied as elsewhere (pars. 14, 15, and 51).

c. Arrest of hemorrhage.—(1) Moderate hemorrhage from a wound about the jaw can usually be checked by pressure from a gauze pack inserted in the wound and held in place by a 4-tailed bandage; the latter also gives some temporary support to a fractured mandible.

(2) Care must be exercised in the application of the pack and the bandage so as not to increase any respiratory difficulty occasioned by the nature of the wound itself.

(3) Hemorrhage that cannot be checked in this way demands a search for the bleeding vessel and application of a clamp to it, followed by ligation, if ligature material is available; otherwise the clamp should be left on during transportation to the hospital.

d. Provision of adequate respiratory airway.—(1) Loss of bone and muscle attachment frequently results in loss of control of the tongue, with danger to adequate respiration, particularly in an unconscious patient. This is best controlled by the use of a long suture through the tip of the tongue, which, in the absence of a needle, may be transfixed with a safety pin; the suture, to which may be tied a piece of gauze or bandage, is used to draw the tongue forward, thus clearing the air passage.

(2) In cases of obstruction due to swelling of the soft tissues, sufficient airway can be provided by the insertion of a rubber tube through the nose or the mouth to the nasopharynx (par. 16).

(3) Tracheotomy should be considered only as a last resort.

e. Temporary approximate reduction and fixation of bone fragments.—(1) If a dental surgeon is available, he should be assigned the problem of temporary fixation; he is provided with an emergency maxillofacial kit, which contains instruments and materials for emergency dental operations and for application of temporary fixation of fractures of the jaws.

(2) Early treatment should be such as to assure every chance for the restoration of original occlusion of the teeth, or the restoration of the function of mastication, even in those cases with considerable loss of bone; it is particularly important that collapse of the bone segments be avoided.

(3) The wound should be cleansed (par. 3), and all tooth fragments, foreign matter, and detached particles of bone should be removed, since these are elements that invite infection.

(4) *Bone particles that still possess periosteal attachment should never be removed, since this small living attachment may make all the difference between new bone formation with restored function, and collapsed fragments with the attendant complications;* even comminuted viable bone should be saved.

(5) Reduction and fixation can only be controlled by skillful manipulation of the segments and the application of simple measures by means of elastic traction and special bandages.

(6) Wiring of teeth of the same jaw across the line of fracture may be used in some cases to maintain fragments during evacuation, but *fixation of the lower to the upper teeth should never be used prior to unattended travel.*

(7) Fixation should be accomplished as soon as possible, since it helps to reduce pain and shock, assists in the control

of the tissues essential for the maintenance of a clear air passage, and is necessary to avoid recurrent hemorrhage and to facilitate recovery; some of these fixation measures may be applied by the dental officer at an advanced station, but must usually be deferred until the wounded man reaches a hospital.

(8) Fractures of the superior maxilla frequently displace the loose structures downward and backward and definitely interfere with respiration.

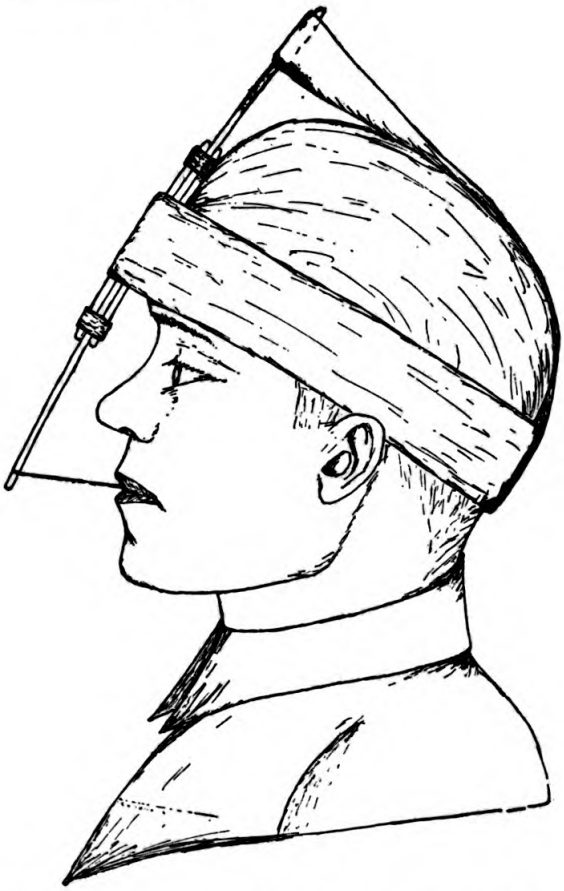

FIGURE 1.—Emergency splint of wooden tongue depressors, bandage, and adhesive tape for holding jaw forward.

(9) With bilateral comminuted fractures of the posterior part of the mandible, the anterior part of the jaw may drop backward and likewise cause serious interference with respira-

tion, in which case the front of the jaw may be held forward by a simple emergency splint (fig. 1).

(*a*) *Material.*

Wooden tongue depressors, four.
Adhesive tape.
Bandage, 2-inch.
Ligature wire (supplied with emergency maxillofacial kit).

(*b*) *Construction.*—Two tongue depressors are placed end to end and are held by two others overlapping them in the middle, all being bound with adhesive tape.

(*c*) *Application.*

1. This is secured with a bandage vertically in the frontal region, the lower end extending in front of the chin.

2. A wire is passed around the lower front teeth or around the chin segment of the mandible, and the ends of the wire fastened to the lower end of the tongue depressor piece, either directly or with a rubber band.

3. The spring of the tongue depressor piece or the elastic effectively keeps the anterior segment of the mandible forward.

In cases of backward displacement of the upper jaw, forward traction can be obtained by attachment of the upper front teeth to this apparatus.

f. Provisions of safe transportation to hospital.—Transportation or evacuation from advanced stations places a certain responsibility on the medical department units, for casualties must be prepared for safe travel by ambulance or hospital train to a hospital.

(1) Nourishment, sedation, means for the prevention of further shock, comfort, and safety are essentials that medical attention must secure for the greatest number.

(2) *Ambulant or semiambulant patients with oral or pharyngeal wounds should sit up;* if they must be recumbent, they should be placed face down if there is any danger of obstruction of the air passages.

8. Wounds of chest.—*a. General.*—(1) The first-aid management of thoracic injuries is concerned with the arrest of hemorrhage from the chest wall and the physical correction of disturbances of cardiorespiratory physiology. The arrest of

hemorrhage is carried out according to established principles of general surgery and requires little comment; the management of disturbances of the function of the heart and lungs requires a basic understanding of the dynamics of the chest.

(2) The risks of prolonged transportation and of early operation should be carefully considered and weighed against each other. It should be remembered that many wounded with apparently desperate injuries recover under conservative supportive treatment, and that early open thoracotomy in the hands of an inexperienced surgeon, with inadequate assistance (untrained anesthetist) or inadequate instruments, may be extremely hazardous and difficult. Early intervention, by an open pneumothorax or by pressure pneumothorax, is urgently indicated by manifestly progressive uncontrollable bleeding. Early control of infection, while desirable, is less imperative and should not impel an inadequately equipped medical officer to operate. Evacuation difficulties may indicate operation, but when easy transportation is at hand, operation should be put off until an adequately equipped hospital is reached.

(3) An opening in the chest wall results in a sucking wound that requires immediate airtight closure. Air accumulating under pressure in the pleural cavity, often forcing its way into the tissues as mediastinal or subcutaneous emphysema, must be afforded a vent. Extensive fractures of the ribs or sternum or defects of three ribs or more following wound débridement result in paradoxical motion of the mobile chest wall and demand stabilization by secure strapping or external fixation. Progressive hemorrhage into the pleural cavity from a lacerated lung is handled by aspiration and artificial pneumothorax. Progressive hemorrhage into the pericardium from a wound of the heart results in cardiac tamponade, which may be relieved by needle aspiration until the necessity of cardiorrhaphy is demonstrated and facilities are available for carrying out the procedure.

(4) Patients with severe mechanical disturbances of respiration suffer from asphyxia, and oxygen therapy should be instituted at the earliest moment. Morphine may make respiratory movements more effective by relieving pain, but if carried to the point of respiratory depression is deleterious.

(5) Definitive surgical treatment of intrathoracic injuries is best carried out with intratracheal anesthesia to provide differential pressure. Anesthetic agents that may be combined

with a high percentage of oxygen (ether) is preferable to agents that may be attended by anoxia (nitrous oxide).

(6) A proper débridement of a thoracic wound may include removal of shell fragments from the lung, resection of devitalized or bleeding pulmonary tissue, hemostatic suture of the lacerated lung, airtight closure of divided bronchi, removal of foreign material and devitalized tissue from the pleural cavity and chest wall, and reexpansion of the normal lung tissues, followed by airtight closure of the chest wall.

(7) There are two important considerations that determine the employment of drainage of the pleural cavity: the prevention or correction of infection, and the prevention or correction of mechanical embarrassment of respiration.

(*a*) Open drainage, which allows free entrance of air into the pleural cavity at the time of operation or subsequently, is employed only for encapsulated collections of pus, with firm adhesions between the rest of the lung and the chest wall.

(*b*) Closed drainage prevents the entrance of air into the chest but allows the escape of fluid and air. It is established by an intercostal tube (usually a catheter introduced through a trocar).

(*c*) Constant closed drainage is provided by attaching a flap valve (condom or finger cot) to the end of the catheter, or connecting the catheter to a longer tube, the end of which is immersed in water. A trap for accumulation of exudate is conveniently placed between the chest and the water-seal valve; the suction of the chest will draw in the water unless this trap is provided or unless the water valve is placed at least 3 feet below the point where the tube enters the chest.

(*d*) Intermittent closed drainage is accomplished by keeping a clamp on the catheter except when an aspirating syringe is being used.

(*e*) Constant closed drainage with suction or closed drainage with intermittent irrigation and suction may be applicable in specialized situations, but for practical purposes can only be carried out in well-equipped hospitals.

(8) Irrigation of drainage tubes is employed only to maintain their patency, and for this purpose warm saline solution should be used. Irrigation of the pleural cavity with chemical antiseptics is strongly condemned except in certain cases of chronic

empyema; chemical antiseptics are *never* to be employed when a fistula exists between the lung and the pleural space.

(9) Drainage tubes must be anchored securely to the external chest wall to avoid their retraction into the pleural cavity. When a tube is draining an area of established infection it cannot safely be removed until the cavity it drains has become obliterated.

(10) Certain general methods, such as the relief of pain (also see above), the treatment of shock, and the administration of chemotherapeutic and prophylactic agents are adequately dealt with elsewhere (pars. 14, 15, and 51).

b. Treatment.—(1) *Tangential or nonperforating wounds without hemoptysis, effusion, or shock.*—(*a*) If the wound is from a bullet, apply a first-aid dressing.

(*b*) If the wound is from a shell fragment, also débride.

(2) *Tangential or nonperforating wounds with hemoptysis.*—(*a*) If the wound is from a bullet, apply a first-aid dressing and a circular bandage, with or without débridement.

(*b*) If the wound is from a shell fragment, also débride.

(*c*) If hemoptysis is severe or persistent, also perform an artificial pneumothorax.

(3) *Tangential or nonperforating wounds with simple, noncompounded rib fractures* (single or multiple).—(*a*) If the wound is from a bullet, apply a first-aid dressing and a circular bandage, eventually replacing with a circular elastic bandage (not too tight).

(*b*) If the wound is from a shell fragment, also débride.

(4) *Compression injuries with traumatic asphyxia or cyanosis involving head, neck, and shoulder girdle.*—These are due to sudden compression of the thoracic viscera, with sudden expulsion of the blood from the great veins and right auricle into the valveless jugular and subclavian veins. Sudden compression of the thorax is usually the result of cave-ins, explosions, and automobile accidents. The head, neck, and shoulders may be deep purple and ecchymotic, the eyes may protrude, the tongue and lips may be dusky and swollen. There are usually multiple ecchymoses and petechiae, and there may be coma from intracranial hemorrhage, or choked optic disks with blindness. Recovery from coma may be protracted. If the patient survives the first day, he usually recovers spontaneously.

(*a*) Provide rest.

(*b*) For stertorous breathing, give 0.3 gram (5 grains) of caffeine sodium benzoate by mouth, or 0.2 gram (3 grains) subcutaneously.

(*c*) For asphyxia, give oxygen inhalations.

(*d*) Examine for ruptured abdominal viscera.

(5) *Extensive mobilization of chest wall due to rib fractures.*—Very extensive unilateral rib fractures may produce serious respiratory embarrassment because of the paradoxical movement of the mobilized wall, plus shifting of the mediastinum. In extensive bilateral fractures, adequate expansion of the lung may be impossible.

(*a*) Provide rest.

(*b*) Give oxygen inhalations.

(*c*) Stabilize and elevate the chest wall by two pairs of perichrondrial wire sutures or of towel clips applied to the 3d and 5th rib cartilages and attached to an overhead frame or plaster jacket.

(6) *Massive atelectasis.*—Massive collapse may involve the injured or, more rarely, the contralateral lung, following blunt or perforating chest injuries, with or without rib fracture. It produces dyspnea, a fall in blood pressure, and, sometimes, cyanosis. Dullness is associated with distant or absent breath sounds (often over the whole hemithorax, whereas in the presence of fluid these signs are found over only the dependent parts of the chest). The hemithorax is immobile, the diaphragm is elevated, and there is displacement of the heart (apex beat) and mediastinum *toward* the affected side. It is due to causes which prevent circulation of air in all or part of one lung, such as splinting of the chest wall and diaphragm from pain, aspiration of blood, mucus, or a foreign body, and compression of one of the main bronchi, but which leave the circulation of blood through the lung intact. The blood absorbs the air, and since no more air can enter, the lung assumes the characteristics of a solid organ.

(*a*) Place the patient on the uninjured side.

(*b*) Encourage coughing and deep breathing.

(*c*) Give morphine, if at all, only in small amounts to control pain.

(*d*) If the above maneuvers are not successful, bronchoscopy and aspiration may be indicated, if feasible.

(7) *Penetrating wounds without serious hemorrhage or shock.*—(*a*) If the wound is from a bullet, apply a first-aid dressing.

(*b*) If the wound is from a shell fragment, also débride.

(*c*) If time and conditions permit, the patient may be X-rayed, followed by a thoracotomy, with removal of the foreign body.

(8) *Penetrating wounds with shock and hematothorax due to injuries of the intercostal vessels.*—This condition is to be expected if signs of hematothorax are present without hemoptysis (see above).

(*a*) Apply a first-aid dressing or, if the wound is wide and open, a ligature or pericostal suture.

(*b*) Place the patient on the injured side.

(*c*) Eventually débride and ligate the ruptured vessels, with rib resection, if necessary, or pericostal suture.

(*d*) Aspirate the blood with a trocar-catheter; *if treatment for shock is necessary and blood or plasma is not available for injection*, the aspirated blood, in the absence of gross contamination, may be reinjected after citrating (one-tenth volume of sterile 2.5 percent aqueous solution of sodium citrate) and filtering through sterile gauze.

(*e*) Close the chest without drainage, approximating the soft parts loosely with interrupted sutures.

(9) *Penetrating wounds with shock and hematothorax due to injuries of internal mammary artery.*—This is a dangerous injury, and exsanguination may take place rapidly (in 2 or 3 hours or less). It is to be suspected if the course of missile traverses the site of the artery (½ inch lateral to the sternal border), if bright-red blood escapes from the wound, and if signs of hematothorax are present without hemoptysis (see above).

(*a*) Both ends of the artery should be ligated as soon as circumstances permit.

(*b*) Aspirate the blood; it may be reinjected after citrating and filtering (see above).

(*c*) Close the chest wall with well-spaced interrupted sutures.

(*d*) Place the patient on the injured side.

(10) *Penetrating wounds with shock and hematothorax from laceration of lung.*—This is to be suspected if signs of hematothorax are present and the patient expectorates blood. The latter sign is not infallible, for bloody expectoration may be absent if the periphery of the lung is torn; on the other hand,

blood may be expectorated from a contusion of the lung without laceration.

(*a*) Apply a first-aid dressing.

(*b*) Place the patient on the injured side.

(*c*) X-ray.

(*d*) If the wound is from a bullet, and the hematothorax is small, provide bed rest and morphine.

(*e*) If the wound is from a bullet, and the hematothorax is large (to the 6th rib or above), aspirate the blood, reinjecting, if necessary, after citrating and filtering (see above), and then perform a pneumothorax.

(*f*) If the wound is from a shell fragment, and the hematothorax is small, débride and treat as with bullet wound.

(*g*) If the wound is from a shell fragment, and the hematothorax is large, débride, remove the blood, and perform a thoracotomy, with extraction of the fragment, débridement of intrathoracic tissues (with suture of the lung to the chest wall and closure of the chest over the packing), or partial resection of the lung (with closure of the chest wall leaving a pneumothorax, or catheter drainage); reinject the blood, if necessary (see above).

(*h*) Keep the patient on the injured side, if possible.

(11) *Penetrating wounds with shock and hematothorax from heart wounds with communication between the pericardial and pleural cavities.*—This is to be expected if there is massive intrapleural hemorrhage with churning sounds over the heart; patients rarely survive.

(*a*) Immediately aspirate the blood; it may be reinjected, if necessary (see above).

(*b*) Repeat if there is a recurrence of hematothorax.

(*c*) If the wound is from a bullet, perform a cardiorrhaphy through a transpleural exposure.

(*d*) If the wound is from a shell fragment, also débride, with removal of the foreign body.

(12) *Penetrating wounds involving heart, with tamponade.*—This is characterized by circulatory collapse, distended external jugular veins, and an immobile cardiac shadow.

(*a*) Aspirate the blood from the pericardium by the costoxiphoid route, if possible.

(*b*) Repeat if there is a recurrence.

(*c*) If it again recurs, perform a cardiorrhaphy through an extrapleural exposure.

(13) *Penetrating wounds with loss of substance of chest wall.*—(*a*) Immediately close the wound by suture, aided by adhesive tape or packing, if necessary.

(*b*) Keep the patient on the injured side.

(*c*) X-ray.

(*d*) Débride, with removal of foreign bodies.

(*e*) Suture the lung to the chest wall and close over a gauze pack, or resect the lung and institute closed catheter drainage.

(14) *Penetrating wounds with pressure pneumothorax.*—Pressure pneumothorax is a very dangerous complication of penetrating injuries. It may be recognized by labored respiration, increasingly intense dyspnea, together with loud tympanitic hyperresonance at the top of the chest when the patient is seated, or at the anterior portion when he is recumbent, absence of breath sounds on the affected side and deviation of the trachea, apex beat, and mediastinum toward the unaffected side. It is likely to be confused with the hyperresonance of a relaxed lung overlying an effusion; however, sharpened breath sounds or bronchial breathing is heard when hyperresonance is due to a relaxed lung, while with a pneumothorax the breath sounds are diminished or absent.

(*a*) Puncture the pleural cavity in the 2d intercostal space anteriorly, at least 1 inch from the sternal border, with a No. 16 gage, 2-inch needle, which should remain only so long as air escapes under pressure.

(*b*) Apply a first-aid dressing.

(*c*) Place the patient on the injured side.

(*d*) If the wound is from a bullet, and air continues to escape, replace the needle with a trocar-catheter in the same location, with a rubber drainage tube submerged under sterile water in a container placed on the floor beside the bed.

(*e*) If the wound is from a shell fragment, X-ray, débride, and perform a thoracotomy, with extraction of the foreign body and suture of the tear or perforation in the lung. If the perforation is small, close the wound air-tight; if associated with extensive laceration, insert a tube with underwater drainage, or suture the lung to the chest wall and close over packing.

(15) *Penetrating wounds with mediastinal emphysema.*—This infrequent complication may occur with wounds of the

trachea or larger bronchi near the pulmonary hilum. It is recognized by labored, rattling respiration, dyspnea, and engorgement of the veins of the neck and upper extremities. Loud, coarse rales may be heard near the midline, and subcutaneous emphysema in the neck, indicated by crackling when pressure is made with a stethoscope above the sternal notch. Mediastinal emphysema may be accompanied by pressure pneumothorax, which should be treated in accordance with directions in the preceding section.

(*a*) Make a suprasternal incision, if warranted by the dyspnea.

(*b*) Withhold fluids or food by mouth.

(*c*) Débride, making a low collar incision, with severance of the sternocleidal attachment, if necessary for adequate exposure.

(*d*) If the trachea is perforated and repair is not feasible, insert a vaseline- or plain-gauze pack down to the trachea.

(*e*) Feed with a small stomach tube, and parenterally if necessary.

(*f*) Observe for symptoms of mediastinitis (chills and fever, and pain or discomfort on swallowing).

(16) *Penetrating wounds with subcutaneous ("surgical") emphysema.*—Subcutaneous emphysema is disagreeable but rarely dangerous or fatal; it almost always recedes spontaneously. When complicated by pressure pneumothorax, mediastinal emphysema or hematothorax, treatment should be directed to these complications (see above).

(*a*) Apply a circular bandage or manual compression at the site of injury.

(*b*) If the wound is from a bullet and uncomplicated, eventually apply compression with a 1-pound or 2-pound sandbag and administer oxygen; in cases in which the subcutaneous emphysema is persistently progressive, débride to the site of perforation (rib fracture), and suture the skin and soft parts over a vaseline-gauze or iodoform-gauze pack.

(*c*) If the wound is from a shell fragment, débride and perform a thoracotomy, with removal of the foreign body. If the wound is small and contamination slight, suture the perforated lung and close the wound tight; if the wound is extensive or grossly contaminated, suture the lung to the chest wall and close the chest wall over vaseline or iodoform gauze, or insert an under-water drainage tube.

(17) *Perforating wounds with sites of entrance and exit.*—These are treated like other penetrating wounds.

(18) *Wounds and injuries of trachea.*—Isolated bullet and shell-fragment wounds of the trachea are exceedingly rare; incised (stab) wounds and suicidal wounds in insane men are occasionally encountered.

(*a*) Control the hemorrhage and apply a first-aid dressing.

(*b*) If there is danger of asphyxia, enlarge the opening through the skin and soft parts; if necessary, a tracheotomy may be performed, providing a cannula is available.

(*c*) Débride, and aspirate blood from the lungs with a catheter inserted into the tracheal opening.

(*d*) If the trachea is not contused, edematous or swollen, suture it loosely, but *not* the skin or soft parts.

(*e*) If suture is impracticable, insert a tracheal cannula.

(*f*) Allow for free escape of air from the site of perforation.

(19) *Pleuroabdominal (transdiaphragmatic) wounds.*—(*a*) Close the sucking pneumothorax by suture.

(*b*) If the wound is from a bullet, proceed with laparotomy (par. 9).

(*c*) If the wound is from a shell fragment, débride.

(*d*) Suture the diaphragm and crush the phrenic nerve if the chest is wide open and the diaphragmatic injury is extensive.

(*e*) Control the pulmonary hemorrhage by suture or resection, and close the chest (using the diaphragm if necessary), tightly if the wound is clean, or over a gauze pack for the skin and soft parts if there is a possibility of infection.

(*f*) Treat intra-abdominal injuries either via thoracotomy and transdiaphragmatic laparotomy or preferably via a new laparotomy opening.

(*g*) For right-sided injuries with laceration of the liver, pack the subdiaphragmatic space and drain through a posterior or lateral incision, with rib resection if necessary, but not transdiaphragmatically and through the open chest. Close the chest tightly or, if obviously contaminated, drain with an under-water tube. Repair the diaphragm, thus keeping the chest and abdomen separate.

(*h*) Left-sided injuries with injury to stomach or spleen need not be drained. After repairing injuries to abdominal viscera close the abdomen. Drain the chest with a dependent catheter and under-water tube, if grossly contaminated. Repair the

liaphragm and keep abdomen and pleural cavity separate, as in ight-sided injuries.

(20) *Rupture of diaphragm.*—This may accompany crushing ijuries of the chest or abdomen, or compression (automobile) ijuries. It may be suspected if there is complaint of much ft-sided epigastric pain and distress, and if there is a hyper-sonant percussion note at the base of the left lung, with ab-nt or diminished breath sounds. X-ray films taken in the ect position, or better an anteroposterior projection in the ght recumbent position, will give the diagnosis. The hernia nually involves the posterior half of the left diaphragm; the omach, a large part of the colon, the spleen, and much of the nall intestine may lie in the left chest in old hernias. Right-ded hernia is rare, the right diaphragm being protected by he liver. However, either the right or left diaphragm may be rn from its parietal insertion.

(*a*) Provide rest.

(*b*) Withhold food by mouth if there is distension, distress, r vomiting.

(*c*) Repair the rupture with interrupted sutures of chromic atgut, either through a laparotomy or thoracotomy opening; he former is usually preferable in early cases without dense dhesions. If repair is done transpleurally, crush the phrenic erve.

(21) *Wounds and injuries of esophagus.*—These are rare, and ndicated by extensive mediastinal and cervical emphysema. hey are differentiated from tracheal injuries by the escape of ir under pressure only when the patient swallows.

(*a*) Withhold fluids and food by mouth.

(*b*) X-ray, giving a small amount of barium.

(*c*) Perform a posterior mediastinotomy, with repair, if pos-ible; drain.

(*d*) If repair is impossible, insert a small stomach tube, hrough an esophagoscope if necessary.

(*e*) If the tube cannot be passed, perform a gastrostomy.

9. Abdominal wounds.—*a. General.*—(1) The time consumed n handling a patient with a complicated abdominal injury must e considered, and it is not permissible, especially when the situ-tion appears hopeless, to operate on such cases to the exclusion f a greater number of casualties of a less complicated nature.

(2) Death following gunshot and other wounds of the abdomen is the result of hemorrhage, shock, or infection, alone or in combination; whereas nothing but the prompt institution of adequate therapy will combat shock and hemorrhage, it is possible that infection can be greatly minimized or even prevented.

(3) Under *ideal* conditions most individuals with definite or even suspected penetrating wounds of the abdomen should be given the benefit of exploratory laparotomy unless the injury is so extensive as to make surgical intervention or even exploration obviously futile; on the other hand, there are certain cases, notably those having simple, penetrating injuries of the liver, that are probably best treated by conservative or nonoperative methods.

(4) Although it has been contended that penetrating wounds of the abdomen caused by small-sized missiles do not require exploratory laparotomy, operation is usually advisable, since serious hemorrhage due to injury of important blood vessels, as well as the presence of gaping, lacerated wounds of the intestinal wall is always a possibility.

(5) Abdominal pain is particularly likely to be absent in perforations of the large bowel, because formed feces escape less readily into the peritoneal cavity and, hence, peritoneal infection does not occur early.

b. Examination.—(1) In addition to the usual methods of auscultation and palpation of the abdomen, one should perform a rectal examination.

(2) A concentric area of contusion about the wound of entrance usually indicates that the bullet took a straight course and that the viscera underlying it are probably injured.

(3) An area of contusion to the right of the edge of the wound suggests that the missile passed from right to left, and vice versa.

(4) The urine should be carefully examined for the presence of blood, to exclude or to verify a lesion of the genito-urinary tract (see par. 10).

(5) X-ray study is important, since it shows not only foreign bodies, but also free gas in the peritoneal cavity; the absence of gas in no way precludes the possibility of perforation on the intestinal tract.

(6) In wounds involving the upper abdomen the period during which operation can be safely delayed is longer than that in wounds of the lower abdomen. Hemorrhage from penetrating wounds of the liver or spleen tends to be self-limiting and perforation of the hollow viscera are less frequent than in wounds of the lower abdomen.

c. Treatment.—(1) *Preoperative.*—(*a*) Patients should be rapidly moved and carefully handled, and body heat should be preserved.

(*b*) General measures, such as the relief of pain, the treatment of shock, and the administration of prophylactic chemotherapeutic agents should be carried out as described elsewhere (pars. 14, 15, and 51).

(*c*) Catheterization should be performed if the patient is unable to void.

(2) *Operative*—(*a*) The anesthetic of choice is one which gives relaxation without increasing the symptoms of shock. Spinal analgesia produces excellent relaxation, but should not be used in patients with extremly low blood pressures. If spinal anesthesia is employed, whole blood, plasma transfusion, 5 percent dextrose in physiologic saline solution, or lactated Ringer's solution should be injected intravenously during the operation to prevent a fall in blood pressure due to relaxation of the arterial bed.

(*b*) The type of abdominal incision varies greatly and is determined by such factors as the site of wound of entry, the location of the wound of exit, and the position of the bullet or shell fragment; rectus abdominus muscle-splitting incisions or paramedian incisions of ample length generally facilitate operative procedures to the greatest degree.

(*c*) On opening the abdomen, all bleeding points should be controlled. In cases in which there is considerable retroperitoneal hemorrhage, the prognosis is bad because the amount of blood that can be lost in this area and because of the difficulty in securing hemostasis. Bleeding is particularly likely to be found in the mesentery or omentum, but can occur from the intestine, from the stomach, and from the liver and spleen.

(*d*) All organs through which the missile might have passed should be carefully examined as speedily as is consistent with thorough observation. The usual order of examination is small intestine, large intestine, stomach and, finally, the kidneys,

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ureters, bladder, liver, gall bladder, pancreas, and bile ducts.

(*e*) The simplest type of closure of intestinal wounds should be employed; resection should not be resorted to unless absolutely necessary; enterostomy is usually not indicated, because ileus can be effectively prevented postoperatively by the introduction of the double intestinal tube (Wangensteen drainage).

(*f*) Hemorrhage from injuries of the liver can usually be controlled by the use of hot packs or by the application of pieces of lacerated muscle, which tend to produce hemostasis. If pieces of liver have been broken off, all fragments should be removed from the peritoneal cavity.

(*g*) Simple penetrating wounds of the spleen are of slight consequence and require little attention; extensive injuries, however, necessitate splenectomy, and are likely to be associated with thoracic injuries.

(*h*) Injuries of the pancreas are of little practical significance; if they do occur they are usually associated with injuries of the large blood vessels and result in fatal hemorrhage before they can be operated on.

(*i*) Injuries of the mesentery are of importance since they frequently involve its blood vessels and, hence, jeopardize the vascularity of the bowel, either because of injury to the vessels themselves or because of mechanical compression of uninjured vessels by an intramesenteric hematoma. Injuries occurring at the junction of the mesentery with the intestine, especially when associated with a dissecting hematoma, are particularly likely to result in the overlooking of an intestinal perforation. After hemostasis has been secured, it is advisable to close a rent in the mesentery by grasping the edges with forceps and tying around the tissue within the grasp of the forceps rather than by introducing a suture on a needle, because the latter procedure is likely to result in perforation of an obscured vessel with further hemorrhage. Injuries of the great omentum should be corrected by ligation of the bleeding points.

(*j*) Injuries of the major vessels of the abdominal cavity are best treated by ligation. Whereas ligation of the aorta or the inferior vena cava is likely to be followed by circulatory incompetence, this may sometimes be prevented by supplementary alcohol injection of the lumbar sympathetic ganglia, which produces vasodilatation of the collateral channels and maintains the viability of the parts distal to the point of ligation.

(*k*) Peritoneal drains should not be inserted; on the other hand, one rubber tissue drain should be placed just superficial to the peritoneum and another just beneath the rectus fascia, being brought out each end of the wound.

(*l*) The abdomen should be closed with through-and-through sutures of cotton or silk thread or stainless steel or silver wire.

(3) *Postoperative.*—(*a*) Heat should be applied to the abdomen and to the extremities to combat ileus and vascular stagnation.

(*b*) If shock is imminent, the patient should be placed in the head-down position; otherwise, the patient should be placed in the head-up position to allow gravitation of the peritoneal fluid into the cul-de-sac of Douglas.

(*c*) Morphine should be given in large amounts in order to relieve pain, to secure rest, and to improve intestinal tone; generally 0.016 gram (¼ grain) of morphine sulfate should be given hypodermically every 3 or 4 hours, unless the respiratory rate becomes less than 12 per minute.

(*d*) An indwelling duodenal catheter or a double tube with continuous suction (Wangensteen drainage) should be used.

(*e*) Enemas and flushes should *not* be used.

(*f*) When there is peritonitis and a likelihood of ileus, the inhalation of *high* concentrations (95 percent or more) of oxygen is of paramount value, but can be accomplished only with special apparatus, such as a B. L. B. mask.

(*g*) Fluids must be given parenterally, and 5 percent glucose in physiologic saline solution or lactated Ringer's solution may be administered in amounts up to 3,000 cc every 24 hours.

(*h*) The possibility of chloride or plasma-protein depletion should be kept in mind.

(*i*) In cases of peritonitis in which sulfanilamide or one of the other sulfonamides has not been given or has been given in inadequate amounts, 250 cc of an 0.8 percent aqueous solution of sulfanilamide should be given by hypodermoclysis every 6 hours until a blood concentration of 5 to 7 milligrams per 100 cc is attained; thereafter smaller doses may be used, but the drug should be continued, if possible, by mouth, until clinical improvement occurs.

10. Wounds of genito-urinary system.—*a. General.*—The relief of pain, the treatment of shock, and the administration of

chemotherapeutic and prophylactic agents should follow the recommendations given elsewhere (pars. 14, 15, and 51).

b. Kidney.—(1) A urine specimen should be obtained from all patients who have suffered injury or gunshot wounds; if the patient is unable to void, urethral catheterization—unless there is urethral injury—should be carried out. Hematuria is a sign of injury to some part of the urinary tract, and the location of such injury should be determined.

(2) Excretory or retrograde urograms should be obtained, if possible, in all cases of suspected renal injury, unless severe shock or anuria is present.

(3) Except in the presence of massive hemorrhage, surgical intervention should be delayed until the period of shock is over.

(4) Surgical intervention, when indicated, should be performed soon enough to employ conservative methods of renal repair.

(5) Gunshot wounds of the kidney almost always require nephrectomy, and are almost always accompanied by other severe visceral injuries, which should be investigated.

(6) In all abdominal wounds associated with hematuria, the condition of the kidneys should be investigated at the time of operation.

(7) Patients who have suffered a renal injury should be kept in bed for at least 10 days.

c. Ureter.—(1) Wounds of the ureter, although rare, demand early operation.

(2) As an emergency measure, provide extraperitoneal drainage for the extravasated urine.

(3) Pass a ureteral catheter prior to operation in all cases of suspected ureteral injury.

(4) Partial tears of the ureter may be repaired, but with a completely severed ureter, nephrectomy is usually indicated.

(5) If the repair of a completely severed ureter is attempted, urinary drainage above the site of the lesion should *always* be provided.

d. Bladder.—(1) Suprapubic tenderness is a cardinal symptom of bladder injury, and its presence should always be sought, particularly in cases of pelvic fracture.

(2) The presence of bladder rupture may be verified by cystography and cystoscopy, if the necessary facilities are available.

(3) Do not catheterize a patient with suspected bladder injury unless facilities for operation are available.

(4) If the bladder is ruptured, immediate operation is imperative, since the mortality rises rapidly with delay.

(5) Provide adequate prevesical drainage for cases of extraperitoneal rupture.

(6) For intraperitoneal rupture, suture of the opening and suprapubic cystostomy, with drainage are indicated; peritoneal drainage should be avoided.

(7) A cystostomy is indicated in cases with massive intravesicular hemorrhage in which the clots canont be evacuated with an instrument passed through the urethra.

(8) In cases of acute retention due to nerve lesions, a catheter should be inserted, and tidal drainage should be established as soon as possible (par. 4).

e. Urethra.—(1) Injuries to the urethra are divided into two classes—those above and below the urogenital diaphragm—and should be diagnosed accordingly.

(2) Do not attempt to pass a urethral catheter unless facilities for operation are present.

(3) A cystostomy is necessary for drainage if the injury is above the urogenital diaphragm.

(4) Urinary extravasation demands immediate incision and drainage, with adequate diversion of the urinary stream by cystostomy or external urethrotomy.

(5) Scrotal and penile injuries should be treated according to the principles of general surgery; adequate urinary drainage should always be provided.

11. Wounds of large blood vessels.—*a.* Immediate control of hemorrhage from a large vessel is called for when bright-red blood is pouring from a wound. This may be accomplished by—

(1) A tourniquet. Rubber bandage is preferred, with rubber tubing next; any material such as rope or cloth may be used in emergency.

(*a*) If sacrifice of limb is probable, it should be applied close to the wound.

(*b*) If early repair is expected, it should be applied at the root of the limb at a distance from the wound.

(*c*) If long transport must follow, the tourniquet should be released every hour for a few minutes, provided the bleeding

can be controlled by digital pressure during the period of release; if this is impossible, the ruptured vessel should be ligated as soon as possible, to lessen the possibility of amputation.

(*d*) *A notation should always be made on the record accompanying the patient that a tourniquet has been applied.*

(2) Direct control of the artery by digital pressure, at the root of the limb as a rule, but conceivably within the wound itself.

b. Suspect injury to a large blood vessel—

(1) When there is an unusual amount of swelling, discoloration, or pulsation in the region of the wound or injury.

(2) When there is pallor, cyanosis, coldness, and a sense of numbness in the limb below the site of injury.

c. Confirm suspicion of injury to a large blood vessel—

(1) By comparing its arterial pulsations with those of the corresponding artery in the other limb.

(2) By making pressure on the main blood vessel proximal to the injury to discover whether any sign of vital color remains in the tips of the extremity (evidence of collateral circulation).

d. Interim treatment in the presence of a serious blood-vessel injury is as follows:

(1) Heat the individual's body and the root of the injured limb; protect the limb from heat loss by woolen coverings, if possible, but never heat the injured limb itself.

(2) Treat shock according to recommendations given elsewhere (see par. 15).

(3) Inject 0.03 gram (½ grain) papaverine intravenously to overcome spasm of the collateral blood supply.

(4) Perform paravertebral novocain block, if feasible, for the same purpose.

e. Ligation of the injured blood vessel involves the following procedure:

(1) With an open wound, ligate and divide the vessel by direct exposure in the wound itself, the wound being enlarged if necessary, especially in connection with débridement; use an Esmarch bandage to control the bleeding.

(2) When approach to the artery through the wound appears too difficult, expose the artery believed to have been injured so far proximal to the point of injury as to secure a clean, dry field (figs. 2 to 11, incl.). For temporary arrest of the hemor-

rhage, pass a small rubber tubing or tape around the vessel. For permanent ligation, divide the artery between two ligatures, and for additional security transfix both stumps with a suture.

(3) The size of the artery and the state of its wall determine the nature of the proper suture material, which varies from medium and heavy silk to tape (for the iliac artery or aorta).

(4) To aid the establishment of a collateral circulation, divide the arteries, rather than ligate in continuity, and divide the companion veins; this causes peripheral vasodilatation. In addition, the sympathetic nerve supply to the limb may be blocked with protaine, or, if convenient and safe, actually divided.

(5) If an artery is found to be thrombosed, resect the thrombosed segment and the companion vein.

(6) Approach the various great vessels at the points indicated in figures 2 to 11, inclusive, in which the optimum sites for ligation are indicated.

f. Incision, approach and collateral circulation in wounds of certain important arteries.—(1) *Common carotid artery—operative procedure* (fig. 2).—(*a*) Place the patient with the head reclining and the neck turned but not overextended toward the opposite side.

(*b*) Incise transversely at the level of the cricoid cartilage, centering over the palpable carotid pulse. Divide the skin, subcutaneous fat, platysma, and fascia in the same direction.

(*c*) Expose the anterior border of sternocleidomastoid muscle; retract this muscle laterally—the superior belly of the omohyoid forms the inner border of the triangle in which the neurovascular bundle lies.

(*d*) Within the vascular sheath the descending branch of the hypoglossal nerve lies anteriorly, the internal jugular vein next, and the common carotid artery slightly behind the medial to the vein.

(*e*) When tying the large vessels, do not pick up the vagus nerve which lies closely behind them; the sympathetic chain lies outside of the vascular sheath.

(*f*) The common facial vein, entering the internal jugular at the carotid bifurcation, may have to be tied for better exposure.

(*g*) When the common carotid artery is tied, collateral circulation is established through the vertebral and the opposite internal carotid arteries, and retrograde through the external carotid.

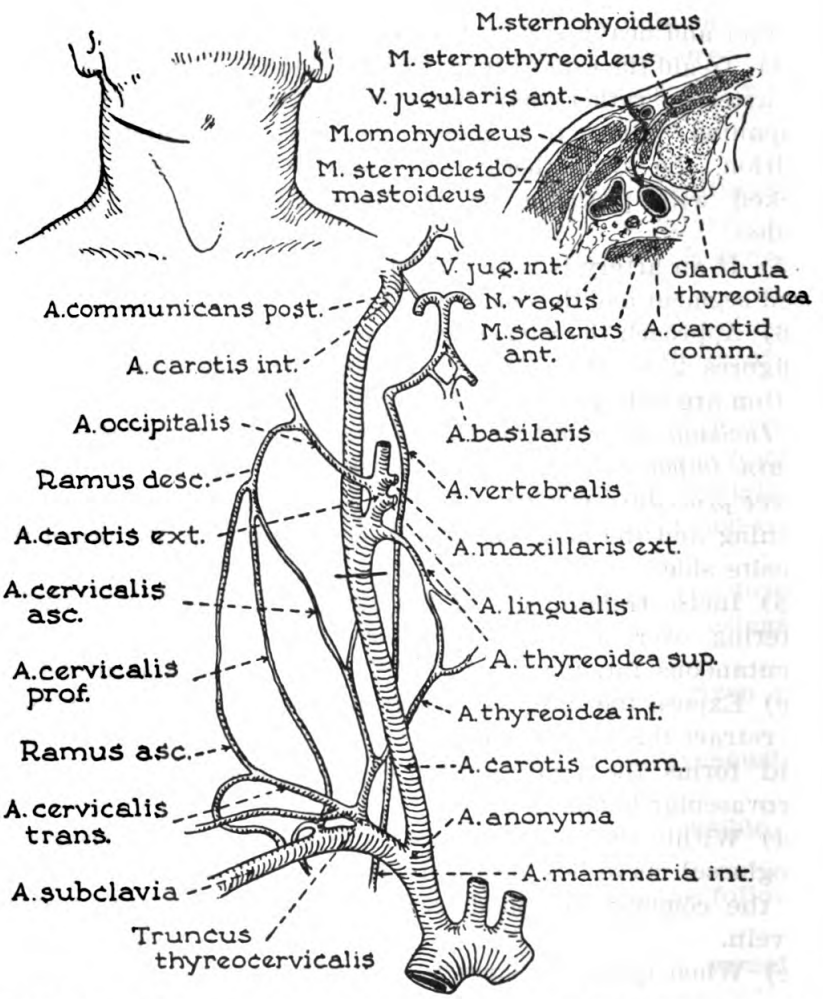


FIGURE 2.—Common carotid artery—incision, approach, and collateral circulation.

(*h*) Avoid trauma to the bifurcation; use procaine to block the nerve receptors in the carotid sinus.

(2) *Subclavian artery—operative procedure* (fig. 3).—(*a*) Recline the head and turn it to the opposite side. Place sandbags under the shoulder blades.

(*b*) Incise parallel to and one fingerwidth above the clavicle over the clavicular insertion of the sternocleidomastoid muscle to the edge of the trapezius.

(*c*) Section skin, platysma muscle, and branches of the supraclavicular nerves.

(*d*) The external jugular vein need not be cut; better ligate between ligatures since nicking it may produce air embolism.

(*e*) Section the sternocleidomastoid muscle to its sternal portion; retract the internal jugular vein medially.

(*f*) Clear the loose fat from the anterior scalenus muscle; define and retract the phrenic nerve medially.

(*g*) Sever the anterior scalenus muscle with its posterior fascia; the subclavian artery lies immediately behind it, over the apex of the pleura; the subclavian vein lies anterior to the scalenus muscle, between it and the clavicle; the brachial plexus lies lateral to the artery, partially covered by the anterior scalenus muscle.

(*h*) Since there is abundant collateral circulation through the thyrocervical trunk and the internal mammary artery, ligate, if possible, distal to these.

(3) *Axillary artery—operative procedure* (fig. 4).—(*a*) Elevate the thorax and moderately abduct the arm.

(*b*) Incise the skin in the groove between the deltoid and major pectoral muscles, beginning at the clavicle for a length of 3 to 4 inches.

(*c*) Retract the edges of the two muscles and identify the transverse fibers of the minor pectoral muscle.

(*d*) Identify and retract the cephalic vein.

(*e*) In the loose fat above the free edge of the minor pectoral muscle, the brachial plexus, more medially the axillary artery and medial to it the axillary vein, are visible. Since ligation proximal to the origin of the two circumflex humeri and the subscapular vessels is safe whereas a tie distal to these may result in gangrene, infraclavicular ligation is preferred to that with the customary axillary exposure, during which the dangerous segment of the axillary artery between the subscapular and circumflex vessels may be tied.

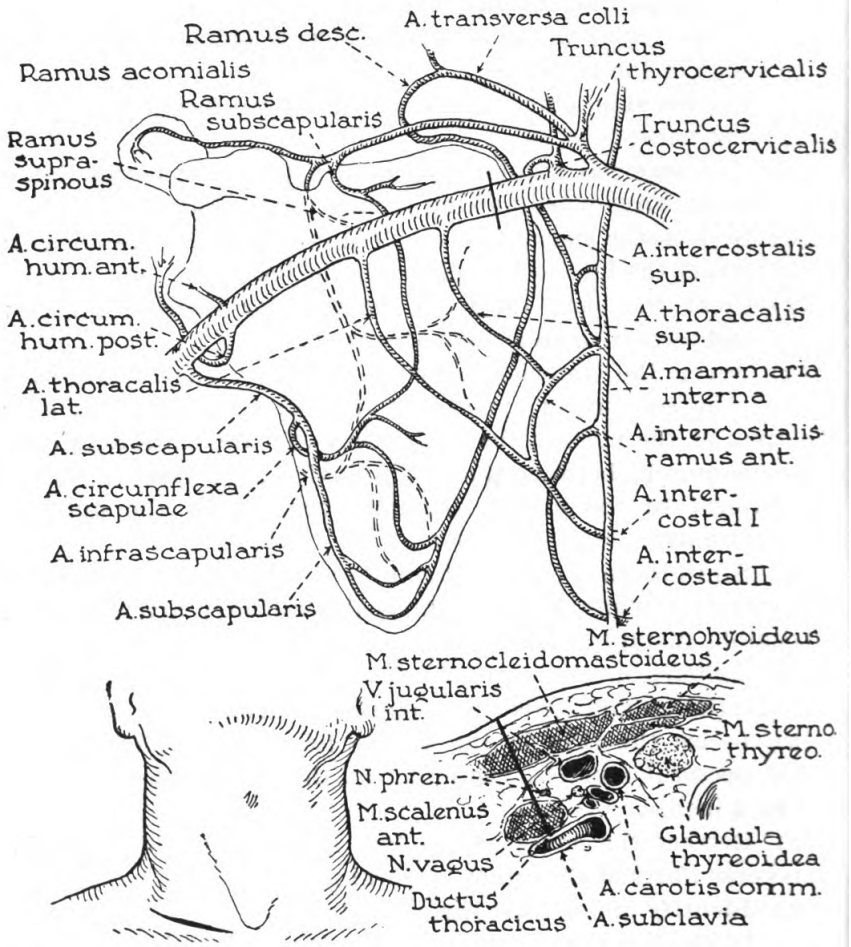


FIGURE 3.—Subclavian artery—incision, approach, and collateral circulation.

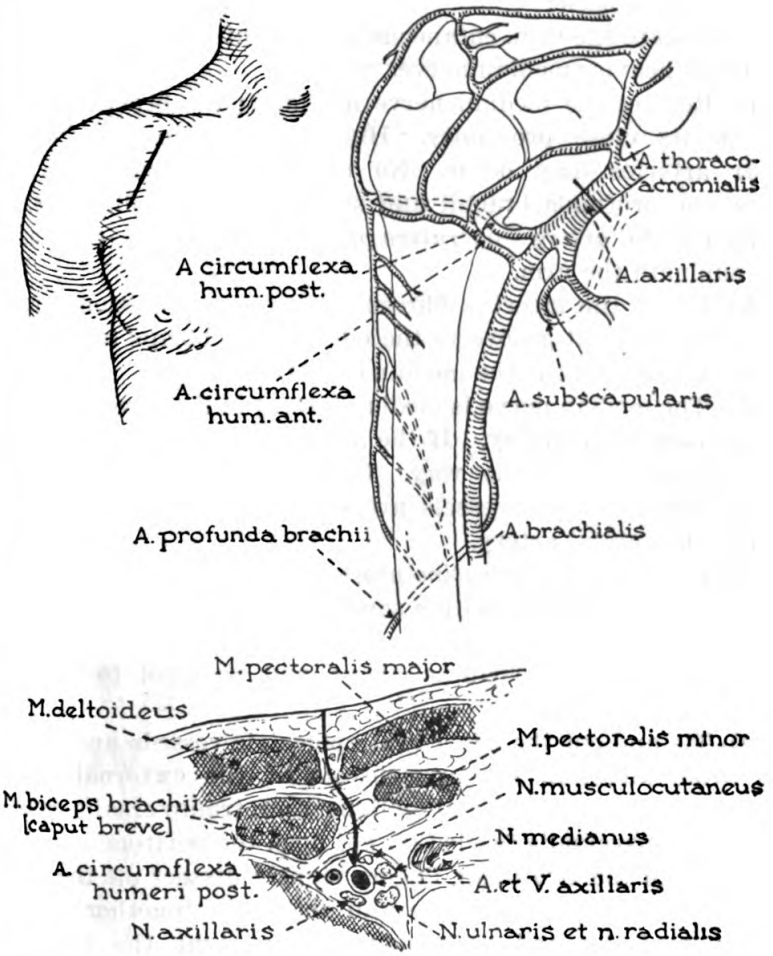


FIGURE 4.—Axillary artery—incision, approach, and collateral circulation.

(4) *Brachial artery—operative procedure* (fig. 5).—(*a*) Abduct the arm in maximal supination.

(*b*) Incise the skin in the middle of upper arm over the bicipital groove.

(*c*) Enter the fascia at the medial edge of the biceps and retract this muscle.

(*d*) Isolate the thin, cutaneous antebrachii medialis nerve and the much heavier median nerve.

(*e*) Retract the median nerve medially; the brachial vessels are in its close proximity. High division into radial and ulnar arteries may occur. No danger accompanies ligation below the profunda brachii and superior ulnar collaterals.

(5) *Cubital artery—operative procedure* (fig. 6).—(*a*) Abduct and supinate the arm.

(*b*) Bisect the lacertus fibrosus through an incision running from the bicipital groove to the edge of the biceps tendon.

(*c*) Ligate and cut the median cubital vein.

(*d*) The median nerve is medial, lateral to it the vein, between and behind it the artery. If the incision is too low the pronator teres muscle is in the way. Collateral supply is abundant unless the recurrent vessels are destroyed below the bifurcation of the cubital artery.

(6) *Iliac artery—operative procedure* (fig. 7).—(*a*) Place the patient flat on his back with a sandbag under the pelvis.

(*b*) Incise the skin from the anterior iliac spine to the pubic tubercle, three fingerwidths above and parallel to Poupart's ligament.

(*c*) Tie and cut the superficial epigastric vessels and sever in the same direction the aponeurosis of the external oblique, cutting across the internal oblique and transversalis muscles and the transversalis fascia; do *not* open the peritoneum.

(*d*) With the two index fingers bluntly dissect off the peritoneum from the psoas muscle and retract it together with the ureter, medially and cephalad; on the right the vena cava divides just behind the common iliac artery; the right common iliac vein first lies lateral to the artery, then passes behind it to its medial side; the left common iliac vein lies altogether medial to the artery.

(*e*) Collateral circulation following ligation of common iliac artery goes through the internal mammary, superior and inferior epigastrics, superior hemorrhoidal, lumbar, and middle sacral arteries; it is insufficient in sudden occlusions but satis-

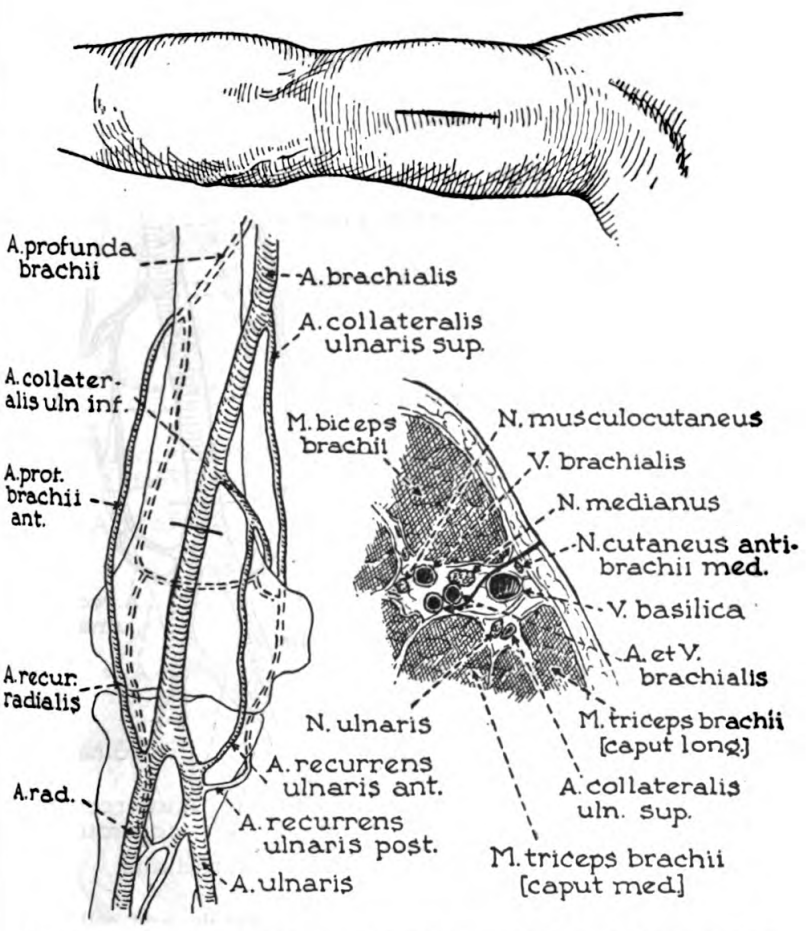


FIGURE 5.—Brachial artery—incision, approach, and collateral circulation.

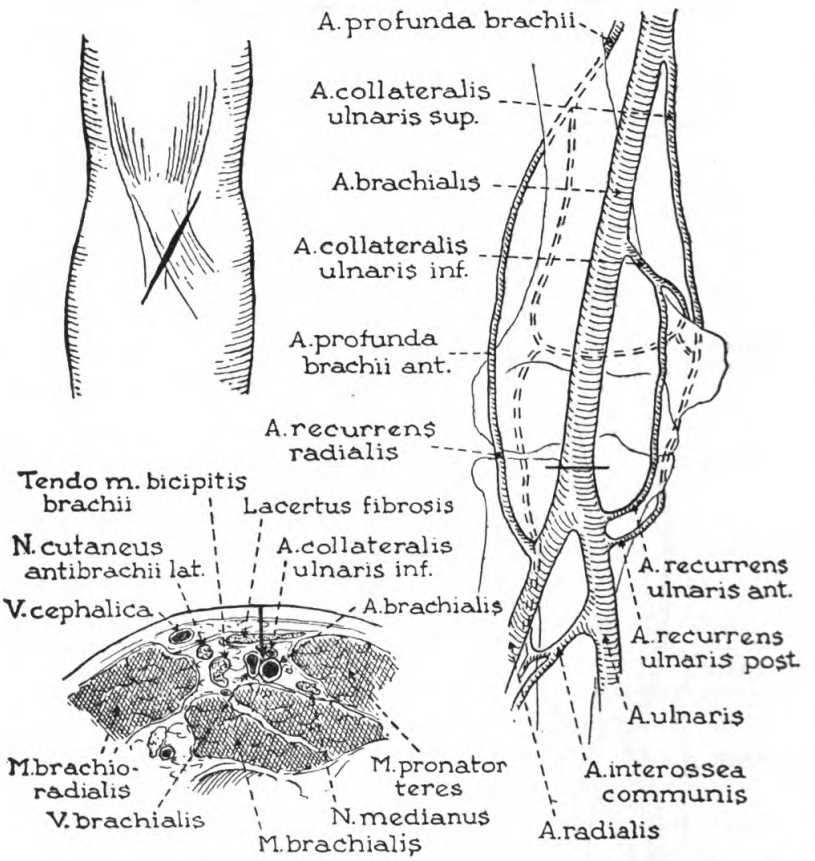


FIGURE 6.—Cubital artery—incision, approach, and collateral circulation.

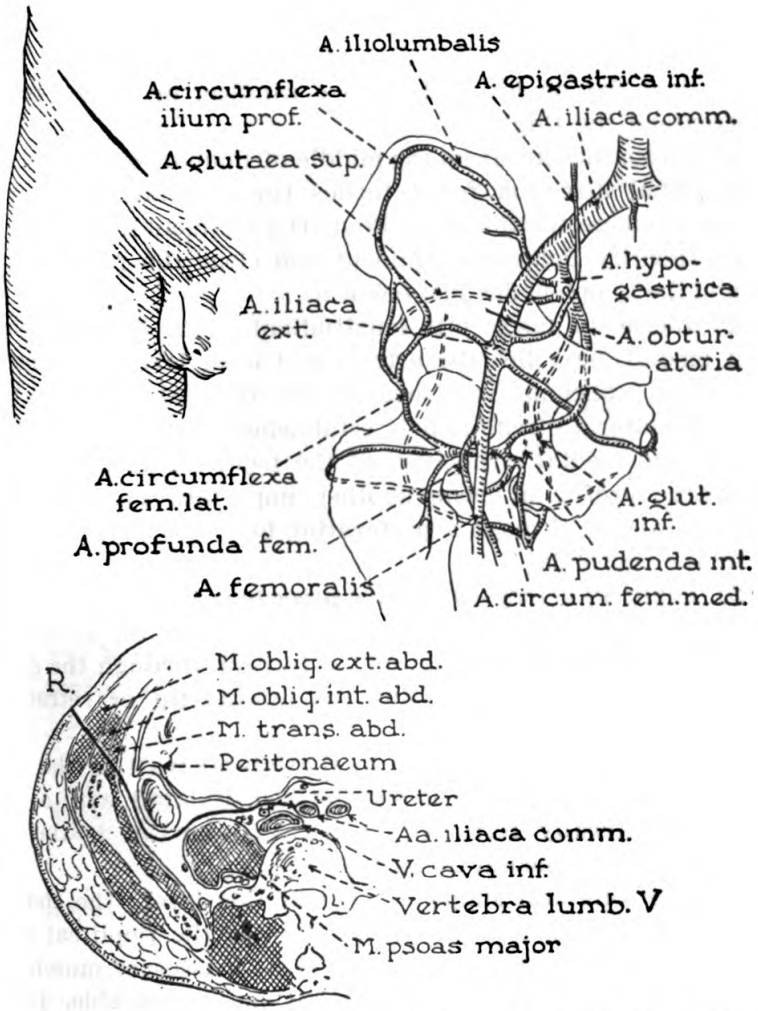


FIGURE 7.—Iliac artery—incision, approach, and collateral circulation.

factory in slowly developing ones or in aneurysms. Collateral circulation after ligation of the external iliac artery is much better. The two important vessels are the circumflex iliac profunda and the deep epigastric as through them the hypogastric artery can feed the femoral, hence the external iliac artery should be tied above the origin of these two vessels.

(7) *Femoral artery—operative procedure* (fig. 8).—(*a*) Place the patient flat on his back with a sandbag or kidney rest to hyperextend the thigh.

(*b*) Incise the skin from the middle of Poupart's ligament in a longitudinal direction for 4 inches (or alternatively, parallel to and two fingerwidths below Poupart's ligament).

(*c*) Identify Poupart's ligament and clear the fascia lata of fat and the lymph nodes just below it.

(*d*) Incise the fascia lata longitudinally; the femoral vein is most medial, next lies the artery, and most lateral the nerve; the major saphenous vein leads to the femoral artery.

(*e*) Collateral circulation is established through the inferior gluteal and medial circumflex to the popliteal artery; ligation below the profunda opens another important channel to the popliteal artery through a perforating branch. Tie, if possible, below the profunda.

(8) *Popliteal artery—operative procedure* (fig. 9).—(*a*) Place the patient on his abdomen with a sandbag under the knee.

(*b*) Make a generous longitudinal incision through the middle of the popliteal space, incising the fascia; tie or retract the small saphenous vein.

(*c*) In the loose fat, closer to the lateral wall of the space (biceps), the tibial nerve is encountered, isolated with a nerve tape and laterally retracted; below and medial is the vein, deepest and most medial the artery.

(*d*) The collateral supply is poor at this level; the channels operating on closure of the femoral enter the popliteal artery above the fossa where there are no large masses of muscle and, hence, small collaterals. If ligation is unavoidable, the tie should be placed above the interior collaterals.

(*e*) Simultaneous vein ligation and sympathetic block are important. Arterial suture is preferable to ligation; this caution is unnecessary in aneurysms of more than 6 weeks' duration.

(9) *Anterior tibial artery—operative procedure* (fig. 10).—(*a*) Place the knee in full extension with the patient on his back.

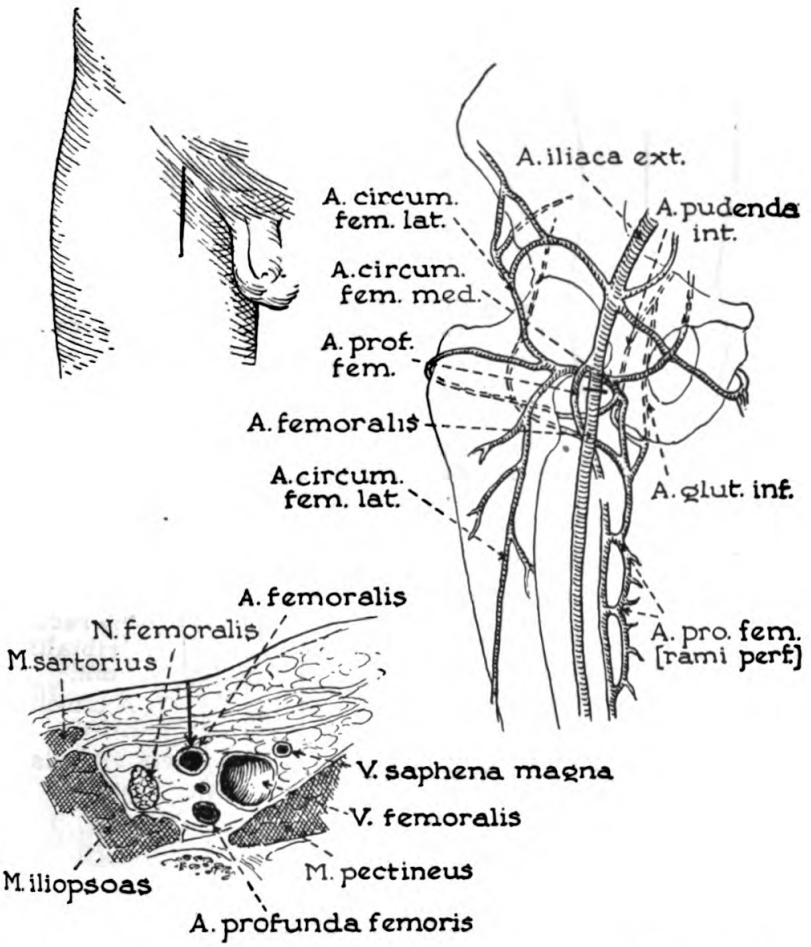


FIGURE 8.—Femoral artery—incision, approach, and collateral circulation.

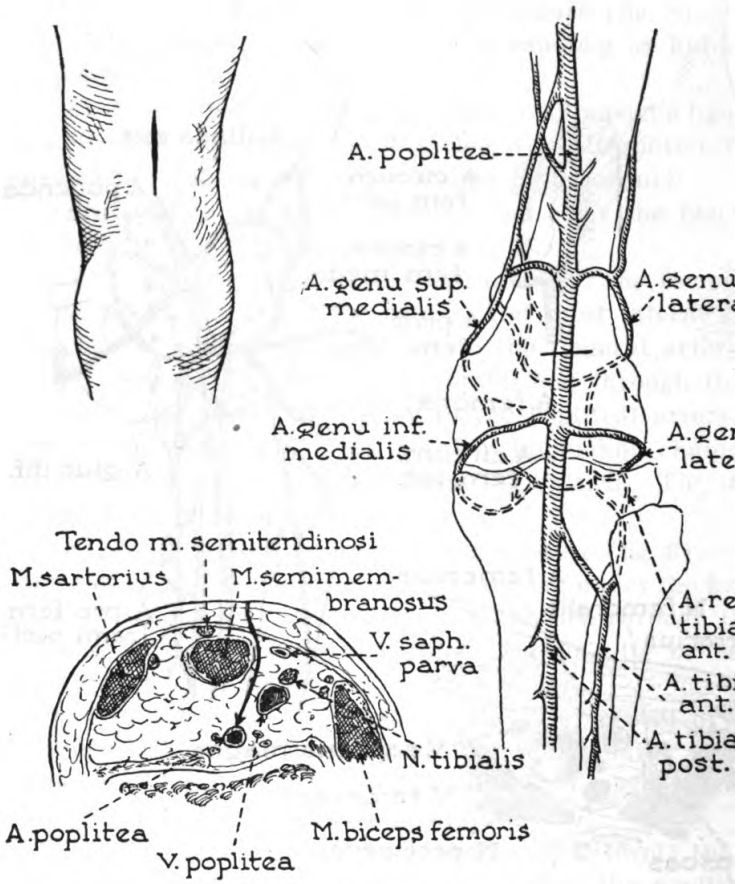


FIGURE 9.—Popliteal artery—incision, approach, and collate circulation.

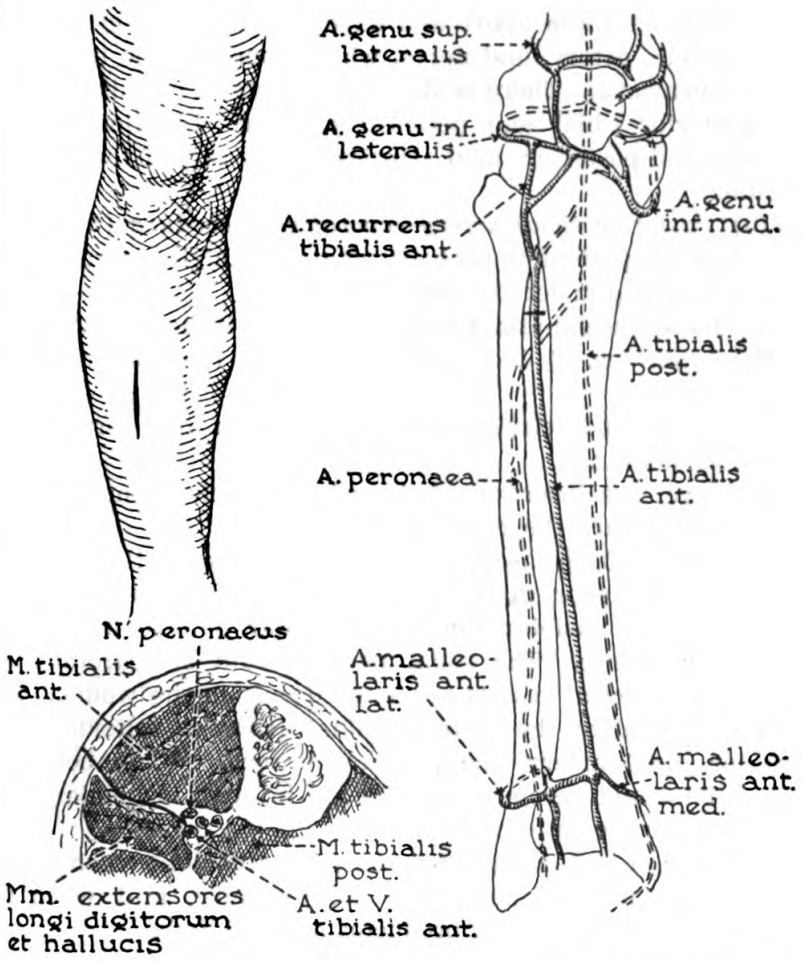


FIGURE 10.—Anterior tibial artery—incision, approach, and collateral circulation.

(*b*) Incise the skin in the middle of the lower leg, a thumb-width lateral to the lateral margin of the tibia, for a distance of 3 to 4 inches.

(*c*) Split the muscle fascia and carefully identify muscle space between the anterior tibial and the long extensor digitorum muscles.

(*d*) Retract these apart and expose, within the cylinder of fat, the vein, artery, and nerve, which approach the tibia from above downward. There is abundant collateral circulation from the posterior tibial and peroneal arteries. Ligation of both anterior and posterior tibial arteries is only safe if the peroneal is intact.

(10) *Posterior tibial artery—operative procedure* (fig. 11).—(*a*) With the patient on his back, rotate the thigh externally and place a sandbag under the bent knee.

(*b*) Incise the skin for a length of about 3 inches in the middle of the lower leg, ½ inch medial from the internal margin of the tibia.

(*c*) Isolate and retract the saphenous vein and nerve in the subcutaneous fat.

(*d*) Sever the insertion of the soleus muscle from the tibial shaft and retract the belly of this muscle.

(*e*) Cut the deep fascia longitudinally; toward the tibia, the fibers of the flexor digitorum longus and the posterior tibial muscle become visible; the neurovascular bundle lies against the latter muscle. The tibial nerve should be carefully isolated; the peroneal vessels are found deeper in the wound. The main collateral of the posterior tibial artery is outside of the anterior tibial artery, the peroneal branch. When both posterior tibial and peroneal arteries are injured, ischemia of the limb may become quite pronounced.

g. Repair of the artery should only be attempted when special suture material and assistance are available, and when the procedure holds promise of producing a useful limb.

h. A pulsating hematoma should not be subjected to emergency treatment, but should be left for deliberate treatment, later.

i. Arterial spasm may be associated with injury to, and especially thrombosis of, the companion vein. It may be due to a blow or a nearby flesh wound, and should be suspected

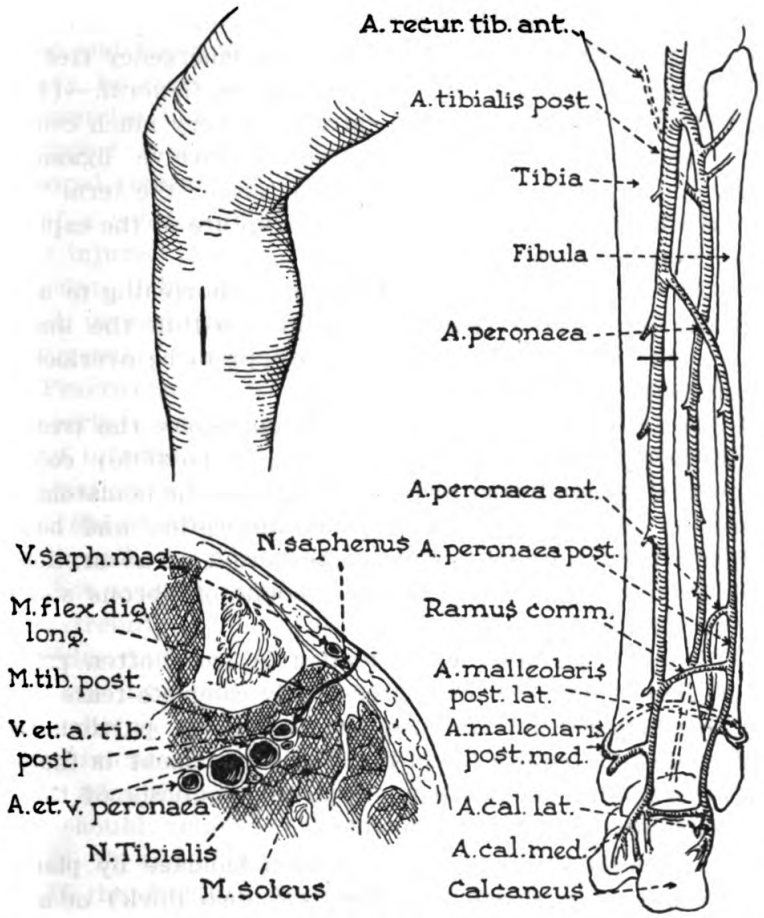


FIGURE 11.—Posterior tibial artery—incision, approach, and collateral circulation.

when the limb is pulseless and cold in the absence of direct evidence of hemorrhage. It should be treated by the application of warmth to the body, the intravenous injection of 0.03 gram (½ grain) papaverine, sympathetic block, or a combination of these measures.

j. Arteriovenous fistula is unlikely to be detected in conditions of emergency, but bruit, thrill, or local arterial pulsation may rarely appear early; it does not call for emergency treatment.

12. Sprains, strains, and contusions.—*a. General.*—(1) The use of the words "sprain" and "strain" is very much confused, but a sprain is usually considered to involve ligamentous tissue, and a strain, muscle or tendon tissue; the term "contusion" means a bruise, with consequent rupture of the capillaries in the involved tissues.

(2) All surgical treatment is aimed at shortening to a minimum the convalescent period—of course within the limits of safety—but this phase of treatment is apt to be overlooked in sprains, strains, and contusions.

(3) In acute sprains, strains, and contusions, the treatment should include (within the first hour, if possible) *cold* and *compression* to minimize the hemorrhage and the hematoma, and to shorten the period of hematoma absorption and healing; such treatment not only reduces the period of absorption but also decreases to a minimum the amount of fibrous scar laid down in the tissues.

(4) A ligament, muscle, or tendon is more often partially rather than completely torn across; only complete tears require operative surgical interference, and all those, particularly in sprains, do not require operation if the treatment is thorough.

b. Treatment.—(1) Make an accurate diagnosis of the ligaments, tendons, and muscles involved.

(2) Immediately apply a compression bandage by placing a properly cut piece of sponge rubber (½ inch thick) or a loose roll of cotton wadding over the region of the injured tissues and holding it in place with an elastic bandage, the whole area being kept cool with ice, if possible; *never* use strapping of adhesive tape.

(3) Rest the injured part for 24 to 48 hours; *never* permit immediate weight bearing in a sprain or strain involving the lower extremities.

(4) During the convalescent treatment, promote recovery and the stimulation of lymphatic absorption of the waste products of the hematoma by the application of heat and by massage, still using the compression bandage as a support between treatments; graduated physical activities may be permitted as healing takes place.

(5) In ligament sprains when tenderness has completely subsided and function has been completely restored, the injured part may be considered healed; then permit participation in active exercise, but only with adequate protection—by adhesive tape support if exercise involves or may involve a strain on the injured tissues.

(6) If a considerable amount of scar tissue is formed in an area of injured tendon or muscle tissue, it will always be a weak point, liable to recurrent injury; when undue strain is expected, protect the previously injured muscles and tendons by a supportive elastic bandage.

13. Fractures.—*a. General.*—(1) Death following fractures occurs early as a result of shock or hemorrhage or late as a result of infection. The dangers of all three are increased by transportation without proper traction splints; hence, the dictum: *Splint where they lie.*

(2) Immediately following a fracture, especially one produced by gunshot wounds, there is a period of paralysis of the muscles of the extremity, which is soon followed by one of spasm, when the muscles contract, thus driving the bony fragments into the soft tissues and thereby causing pain and shock; with early splinting, before the period of spasm has begun, this is avoided.

b. Treatment.—(1) The relief of pain, the treatment of shock, and the administration of chemotherapeutic and prophylactic agents should follow the recommendations given elsewhere (pars. 14, 15, and 51).

(2) If the fracture is compound, the clothing is cut away from the wound, hemorrhage is controlled (par. 11), and an occlusive dressing, a first-aid-packet dressing or a shell-wound dressing, depending on the size of the wound, is applied; make *no* attempt to cleanse or débride the wound.

(3) The extremity is splinted as follows:

(*a*) For fractures of the femur, fractures about the knee joint, or of either or both bones of the leg above the ankle, a hinged half-ring splint is used.

(*b*) For fractures of the upper extremities, including those about the shoulder and elbow, the Murray-Jones traction splint is used.

(*c*) For fractures of the wrist and hand, a padded-board coaptation splint is used.

(*d*) For fractures of the ankle and foot, a wire-ladder splint is used.

(*e*) For fractures of the spine, moderate extension is indicated; this can be accomplished with the patient prone or recumbent, but in the latter position a rolled blanket of pillows *must* be placed under the site of injury (par. 4).

(*f*) For fractures of the neck, improvised support, with moderate extension, is provided.

(*g*) For fractures of the pelvis, a swathe about the pelvis, with the thighs and legs fixed together by bandages, is applied.

(*h*) Detailed information in regard to the construction and application of the splints is contained in FM 8–50.

(4) A fracture or wound in which a joint is involved should be treated like a compound fracture, no attempt being made to cleanse the wound.

(5) The patient is immediately transported to a hospital, care being taken that he is properly covered with blankets, to maintain body heat.

14. Relief of pain.—*a. General.*—(1) Relief of pain following injury is of the utmost importance, not only because it alleviates suffering but also because pain is one of the main contributory factors in surgical shock.

(2) Pain may be relieved in two ways, neither of which should be omitted without cause:

(*a*) First, proper first-aid treatment should be given, if possible, to the injured part; thus, the application of a traction splint to a fractured extremity, of a tight swathe to certain types of chest injury or of a firm supporting bandage to a flesh wound frequently results in complete or partial relief of pain.

(*b*) Secondly, sedatives of the proper sort and in proper amounts should be given whenever indicated, particularly before painful first-aid manipulations or before transportation.

(3) Sedation is accompanied by depression of the heat-regulatory mechanism, hence extra precautions should be taken to protect the sedated patient against the effects of cold.

b. Methods of sedation.—(1) For severe injuries and severe pain, morphine is the drug of choice and should, if possible, be given hypodermically.

(*a*) Inject 0.016 gram (¼ grain) morphine sulfate and repeat according to the amount and duration of the relief experienced.

(*b*) Pain is an antidote for morphine, and a rugged man with an excruciatingly painful injury may require three injections at 15-minute or 30-minute intervals before relief is experienced; however, if the respiratory rate is below 14 per minute, the dose should not be repeated.

(*c*) Morphine should *never* be given to unconscious patients or those with a head injury or with actual or potential respiratory embarrassment.

(2) The barbiturates are particularly useful for moderate degrees of pain associated with fear, emotional distress, or nervousness.

(*a*) Give 0.15 to 0.30 gram (2½ to 5 grains) barbital or phenobarbital by mouth, previously dissolving the drug in a warm fluid, if possible; or, inject 0.15 to 0.30 gram (2½ to 5 grains) barbital sodium or phenobarbital sodium intramuscularly.

(*b*) The dose may be repeated in 3 hours, if necessary.

(*c*) Often the combined use of morphine and a barbiturate is more effective than either one alone; however, full doses should not be repeated as often as when given alone.

(3) For minor degrees of pain, acetylsalicylic acid (aspirin) in 0.6-gram (10-grain) doses every 2 hours, is recommended.

15. Secondary or wound shock.—*a. Mechanism.*—(1) Primary shock refers to a condition of collapse that may follow quickly after the receipt of an injury; it is usually to be explained on a neurogenic (reflex or psychic) basis. Measures that may be of aid in treatment include the recumbent position, the giving of stimulants, the application of heat, the relief of pain, and the use of vasoconstrictor drugs. An early favorable response is usually obtained, unless it is complicated by blood loss or other shock-producing factors. This type of shock is usually of short duration, although it may progress into the secondary type.

(2) (*a*) Secondary or wound shock is usually slower in onset than primary shock and is most apt to develop an hour or longer following injury. The predominant feature is a

marked decrease in circulation, generally produced by the loss of an effective blood volume, which, in war, usually results from hemorrhage. The output of the heart declines before the blood pressure, and hence a marked decline in the blood pressure is more than a danger signal; it indicates a marked impairment of the blood supply to the tissues, with resulting anoxia, which, in turn, produces a general increase of capillary permeability, with loss of plasma from the vessels.

(*b*) The reduction of the blood volume in secondary shock may be due to a loss of whole blood or blood plasma or both. Dehydration, fatigue, pain, fear, exposure to cold, and other factors may contribute to its origin. During the early stages of the development of shock due to the loss of whole blood, dilution of the red blood corpuscles usually results, unless the subject is markedly dehydrated. During the terminal stages of prolonged shock from any cause, even hemorrhage, a general increase in capillary permeability with plasma loss and hemoconcentration may result. Hemoconcentration is observed throughout the early stages of shock, and largely results from a generalized plasma loss into the tissues. A diminution in the blood volume caused by plasma loss is more dangerous than the same reduction due to hemorrhage. The combination of the loss of whole blood and plasma may be such that there is no alteration in the concentration of the red blood corpuscles.

(*c*) There is no infallible, easily performed laboratory procedure which acquaints one with the condition of the circulation in the incipient stage of shock.

b. Clinical picture.—(1) There is no difficulty in the recognition of the *advanced stages* of shock. The findings include pallor, weakness, cold extremities, rapid pulse rate, low blood pressure, sweating, and frequently, vomiting. Unfortunately, treatment is often of no avail when the advanced stages have been reached.

(2) The clinical recognition of the *early stages* is not so easy. Pallor and tachycardia, which are the usual findings in early shock, may be caused by other disturbances. A history of injury in a patient with cold, pale skin and a pulse rate greater than 100, particularly if these abnormalities persist for a number of minutes, usually means incipient shock. Treatment should be instituted immediately, even though the systolic blood pressure is not below 100. It should be remembered that

the arterial pressure is being maintained by vasoconstriction and is likely to decline rapidly with the slightest development of shock. The reduced blood volume, vasoconstriction, and lowered blood pressure cause diminution in tissue circulation, and if this state is continued too long, the oxygen lack causes irreparable damage. Signs of progression of shock include a further increase in pulse rate, a decrease in the pulse pressure, an increase in pallor, and sweating.

c. Treatment.—(1) *General.*—The prevention of fully developed failure of the peripheral circulation is the most important phase of treatment. The patient in profound shock may be unable to recover even if his blood volume is fully restored with blood itself. The clinician should recognize the early signs and should immediately carry out the therapy that is indicated. It cannot be emphasized too strongly that success usually depends on the promptness with which the proper treatment is instituted. It is generally agreed that the best single form of therapy consists in augmenting the reduced blood volume by the introduction of blood or blood substitutes. An estimate should be made of the factors that are contributing to the shock, such as dehydration, continued bleeding, cold, pain, and fear, and various corrective measures should be performed. The treatment that can be given by various medical units is dependent on the facilities that are available and on military operations or enemy action at the time; however, one should always approach the ideal as nearly as possible.

(2) *Arrest of hemorrhage.*—(*a*) This is by far the most important step. The available means for arresting hemorrhage in the very advanced positions in active engagements are usually limited to digital pressure on the artery between the wound and the heart, the application of a tight dressing, the insertion of a sterile gauze pack, and the use of a tourniquet (par. 11). A tourniquet should usually be employed only when other means for controlling hemorrhage are inadequate. There is no objection to placing a tourniquet on an extremity that is injured beyond repair. With civilian casualties, where it is known that the constriction can soon be released, the tourniquet should find a more extensive field of usefulness than in field operations. A fractured limb should be splinted during transport. This lessens the additional loss of blood and plasma at the site of injury and reduces the severity of the pain.

(*b*) When the casualty has been transported to a place where adequate facilities are available, it should be noted whether or not the bleeding has stopped. If it continues, isolation of the blood vessel and its ligation and division may be necessary (par. 11). If a tourniquet is in place and an amputation is to be performed, the tourniquet should not be removed until the time of or after the amputation. Preliminary removal of the tourniquet is followed by the passage of a considerable part of the blood volume into the dilated peripheral blood vessels and by the further loss of whole blood and plasma from the injured vessels. If the tourniquet is removed and an attempt to save the part is to be made, facilities for blood transfusion should be available.

(3) *Relief of pain.*—There is convincing evidence that morphine in doses of 0.016 to 0.032 gram (¼ to ½ grain) is of aid in the prevention of shock. Larger amounts or doses at too frequent intervals may result in depression of the respiratory center. It should be given for the pain and restlessness associated with injuries, except all intracranial ones, before the patient is moved from the scene, and is preferably injected subcutaneously or intramuscularly, although it may be given by mouth. Sodium phenobarbital in doses of from 0.18 to 0.30 gram (3 to 5 grains) parenterally should be given for the restlessness associated with intracranial injuries.

(4) *Body temperature.*—(*a*) Every effort should be made to prevent and to correct excessive chilling. Stretcher bearers should be familiar with the correct use of waterproof sheets and blankets. When blankets are not available, protective clothing should be placed between the canvas of the stretcher and the patient. More protection is needed under the body than over it. Wet clothing should be removed and dry garments should be substituted as early as possible.

(*b*) When facilities are available, the chilled patient should be warmed by artificial means. This may be accomplished by hot-water bottles, by warm bricks or stones wrapped in blankets, or by more elaborate means, such as a heat cradle or an electric blanket. Unless contraindications exist, warm fluids may be given by mouth or by rectum.

(*c*) It should be realized that excessive heat may cause as severe ill effects as excessive cold. Peripheral vasodilation due to overheating may cause a decrease of the blood flow to the more vital structures.

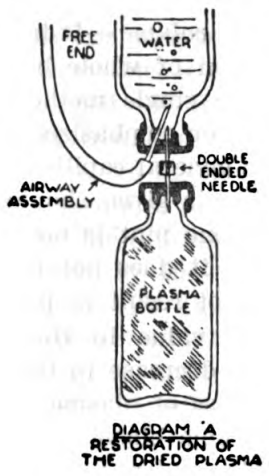
(5) *Restoration of blood volume.*—It is generally agreed that the intravenous introduction of whole blood and blood substitutes is the most valuable single method for preventing and combating shock. It has been emphasized that the blood volume should be restored before general capillary damage takes place. The quantity of blood that is given should be governed by the needs of the patient. If one pint is not effective in restoring an effective blood volume, it does not follow necessarily that three or four pints will not result in permanent benefit.

(*a*) Whole blood is of value in the treatment of shock, regardless of whether the decrease in the blood volume is due to hemorrhage or to the loss of plasma. There are many features about the use of whole blood that frequently makes its employment impracticable under field conditions. These include difficulties in preservation, in transportation, and in making compatibility tests. There is, however, no valid objection to the use of fresh or preserved (for not more than 8 days) blood, if these obstacles can be overcome.

(*b*) Liquid blood plasma and serum present advantages over whole blood: compatibility tests are not necessary and the keeping qualities are superior. Most cases of shock are associated with hemoconcentration; hence a given quantity of plasma or serum is more effective in treatment than an equal quantity of whole blood. Furthermore, blood plasma and serum are approximately as effective in the treatment of hemorrhage as is whole blood. One objection to the use of these fluids in advanced stations is the fact that ideally they should be stored at icebox temperature.

(*c*) Dried blood plasma and serum (fig. 12) present the advantage, if properly prepared, of prolonged preservability. Denaturation is slow, and the multiplication of bacteria is retarded. Exposure to moisture must be avoided. Compatibility tests are not necessary. One has only to dissolve the powder in distilled water and to introduce the solution intravenously.

(*d*) Isotonic salt and glucose solutions are useful in the treatment of shock when plasma or serum are not available. Restoration of the blood volume, however, is only temporary, since the fluid rapidly leaves the blood stream. Furthermore, the intravenous introduction of large quantities of solutions of crystalloids in the presence of capillary damage may result in the loss of a large amount of protein from the blood vessels.



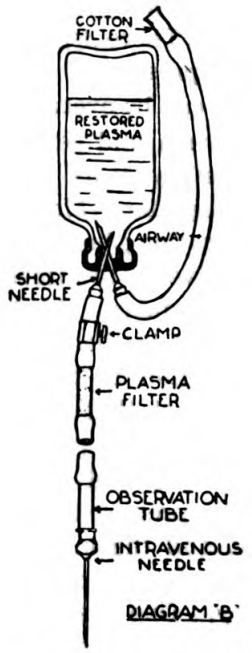


FIGURE 12.—Standard Army and Navy package of dried human plasma.

Instructions for use.—1. Open metal cans with attached keys.

2. Remove plasma and water bottles. Cleanse stoppers with alcohol.

3. Remove cellophane from double-ended needle and remove glass tube from one end of needle.

4. With water bottle in upright position insert uncovered end of double-ended needle through stopper into the water bottle.

5. Remove cellophane and glass tube covering airway needle and in-

sert needle of airway assembly through rubber stopper into the water bottle.

6. Elevate free end of airway assembly to prevent water from wetting cotton filter in airway. *Caution:* If cotton in airway filter becomes wet—remove it.

7. Remove glass tube from other end of double-ended needle, invert water bottle and insert needle through stopper into plasma bottle. (See diagram A.)

8. Allow water to be drawn in plasma bottle.

9. After water is added, double-ended needle is removed from plasma bottle.

10. Shake plasma bottle until plasma is completely dissolved.

11. Apply metal clamp to the 2″ piece of rubber tubing on the intravenous set and close it.

12. Remove coverings from short needle attached to intravenous set and insert through stopper of plasma bottle.

13. Withdraw needle of airway assembly from water bottle and insert through stopper into plasma bottle.

14. Invert plasma bottle and suspend it for administration. (See diagram B.)

15. Fix glass end of the airway assembly with the suspension tape above the inverted plasma bottle.

16. Remove cellophane from observation tube and intravenous needle.

17. Attach intravenous needle to tube and remove glass tube from needle.

18. Loosen metal clamp and allow plasma to fill rubber tubing. When tube is filled, tighten metal clamp.

19. Insert needle in vein and regulate flow with screw clamp. If patient is to receive additional plasma, restore second bottle as outlined. Close regulating clamp as soon as first bottle is empty, but before air enters tube. Pull out needles from first bottle and insert in second bottle. Elevate end of airway and fix it in place with the suspension tape. *Caution:* If vacuum in plasma bottle is lost, apply pressure in water bottle by forcing air into airway tube. If this method fails, remove stoppers and pour water into plasma bottle. Replace stopper on plasma bottle and administer immediately.

It is in the correction of conditions that may *lead* to shock, such as dehydration, that these solutions find their greatest usefulness.

(*e*) Hypertonic solutions of crystalloids are rarely indicated in the prevention and treatment of shock.

(*f*) Gum acacia in saline solution may be effective, but it is much more dangerous than plasma or serum and probably should not be used.

(6) *Correction of dehydration.*—Dehydration is one of the most frequently encountered contributing agents in the devel-

opment of shock. Since the loss of water is accompanied by the loss of salts, fluids that are given by mouth or otherwise should contain salt (drinks should contain half a teaspoonful of salt to the pint). Unfortunately, many injured patients are unable to retain fluids when given by mouth, and other routes are therefore frequently necessary. If the condition of the circulation is such that fluid can be absorbed from the subcutaneous tissues or rectum, these routes are preferable to intravenous injection. If intravenous therapy is necessary, it is well to alternate the giving of isotonic saline solution with that of whole blood or plasma. As stated previously, the intravenous injection of large quantities of solutions of crystalloids may result in damage. Excess salt solution may cause edema, and 5 percent glucose solution should be alternated with or mixed with saline, particularly if edema is present.

(7) *Oxygen inhalations.*—Tissue anoxia occurs in shock and is somewhat relieved by the inhalation of a high concentration of oxygen (95 percent or higher). As a matter of fact, the benefit is not striking, the apparatus is cumbersome, and usually one's efforts had better be expended in some other form of therapy.

(8) *Vasoconstrictor drugs.*—Vasoconstriction is usually present in secondary shock except in the terminal stages, and the production of additional vasoconstriction may result in harm rather than benefit. An increase in the blood volume and in the effective flow of blood to the tissues, rather than solely an increase in the blood pressure, are to be desired, and these changes can be accomplished most satisfactorily by the introduction of blood or blood substitutes. In primary or neurogenic shock, vasoconstrictor drugs may be given, as previously indicated.

(9) *Adrenocortical extract.*—Potent extracts have been available only a short while, and evidence as to their effectiveness is somewhat controversial.

(10) *Anesthesia.*—(*a*) Whenever possible, local anesthesia should be used for patients in shock. If an inhalation agent is used, it should be combined with an adequate quantity of oxygen, since anoxia is already present. For this reason, oxygen and ether is preferable to oxygen and nitrous oxide, since the latter mixture generally results in cyanosis.

(*b*) General anesthesia should be avoided or postponed,

whenever possible, until the shock has been treated and the patient's condition has improved. Disastrous consequences frequently result from a failure to appreciate the danger of general anesthesia in a critically wounded patient.

16. **Respiratory emergencies.**—*a. General.*—A respiratory emergency is any marked interference with the transport of oxygen to or carbon dioxide from the central nervous system.

b. Mechanism.—It may result from one or more of the following:

(1) Decrease of respiratory exchange.

(2) Obstruction of the upper respiratory passages due to tongue position, edema, or spasm from irritants.

(3) Blocked alveolar absorbing surface caused by blood, water, vomitus, fibrosis, or pneumonitis.

(4) Marked decrease in the rate of flow or quantity of circulating blood.

(5) Interference with the normal negative pressure of the pleural cavity (wounds of the chest wall and diaphragm—pneumothorax).

c. Causes.—The etiologic agents are shock, hemorrhage of the upper respiratory tract, drug overdose, gas poisoning, immersion, vomitus, edema, blood or pus collections pressing on the respiratory tract or rupturing into it, brain injury, paralysis, and wounds of the thoracic wall, viscera, face, or neck.

d. Signs and symptoms.—These are as follows:

(1) Inadequate, labored, noisy, rapid, or dyspneic breathing, or apnea.

(2) Pulse abnormalities.

(3) Pale, ashen-gray, or cyanotic color of the skin.

(4) Mental disturbances (overconfidence, delirium, or unconsciousness).

(5) Muscular disturbances (tremors or convulsions).

e. Treatment.—Attempt to relieve respiratory obstruction and to restore the proper oxygen and carbon dioxide transport as follows:

(1) The patient should be placed laterally with the head down, or prone if fluids or foreign bodies are suspected. The mandible and head should be adjusted so that they are in proper relation to the cranium and thorax, respectively; pull the tongue forward.

(2) If a foreign body is present in the pharynx, larynx, or

trachea, an attempt should be made to remove it. Try a sharp blow on the back, with the body suspended head down, or wiping, or suction, if available. Endotracheal suction with direct vision through a laryngoscope may be tried, if necessary, and if the apparatus is at hand.

(3) It may be necessary to establish an artificial airway. A rubber tube passed down to the larynx or into the trachea through the mouth or nose, either blindly or with an anesthetist's laryngoscope, is often life-saving. In the presence of hemorrhage, a pack should be placed firmly around such a tube. *Remember that a tracheotomy can be performed with a pocketknife.*

(4) In cases due to the inhalation of an irritant gas, with acute laryngospasm and edema, the following emergency treatment should be given:

(*a*) Spray into the upper respiratory tract during attempts at inspiration a 5 percent cocaine solution, nebulized to as fine a cloud as possible.

Caution: It should be remembered that cocaine is extremely toxic to some individuals. A solution of this strength should be used only as an emergency measure, and then only in such amounts as the emergency warrants.

(*b*) Administer the richest oxygen atmosphere obtainable.

(5) If inadequate ventilation is due to drug depression, head injury, open pneumothorax, shock, hemorrhage, or partial obstruction that cannot be removed, oxygen enrichment of the inspired air is beneficial. *Always use it if available.*

(6) (*a*) If exchange is inadequate and oxygen is not available, respiration may be enhanced by—

1. Mouth-to-mouth, mouth-to-nose, or mouth-to-artificial-airway breathing.

2. The Schaefer maneuver (see par. 29).

(*b*) If respiration is absent, attempt to reestablish it by these methods instantly; the airway must be patent (see above).

17. Burns.—*a. General.*—Burns, as discussed in this paragraph, include all cases with damage of the skin and underlying tissues due to heat, chemicals, or electricity.

b. Fundamentals of treatment.—(1) The prevention and control of shock is the primary consideration in the management of every burn.

(2) Proper prophylactic measures against pyogenic infection, tetanus, and gas gangrene should be taken.

(3) Satisfactory end results, that is, minimal scar formation, largely depend on the avoidance of pyogenic infection; the moist surfaces of burns provide ideal conditions for bacterial growth, and it is therefore of paramount importance to employ, if possible, strict aseptic technic in operating on and dressing burns.

c. Treatment.—(1) Proper steps for the prevention or treatment of shock (par. 15) should be instituted.

(2) In all cases with moderate to severe burns, prophylactic chemotherapy should be administered. Sulfadiazine is the drug of choice (sulfanilamide may be substituted), with an initial dose of 2.0 grams (30 grains) and subsequent doses of 0.5 gram (7½ grains) every 4 hours, the small doses being necessitated by the possibility of impaired kidney function. If normal kidney function can be clearly demonstrated, larger doses of sulfadiazine can be given (1.0 gram every 4 hours).

(3) Prophylaxis against tetanus (par. 49) is indicated in all patients with second or third degree burns.

(4) A prophylactic dose of gas-bacillus antitoxin (par. 48) may be given at the discretion of the medical officer.

(5) The burned area should be treated as follows, standard operating room technic, if possible, being employed, with the patient, as well as all attendants, fully masked:

(*a*) The burned area and then, separately, the surrounding skin are carefully cleansed with neutral soap and water; ether or benzene is used to remove grease, if present.

(*b*) All blisters and loose shreds of epidermis are carefully removed, and this material is saved for bacteriologic study, if feasible. Skin that gives evidence of irreparable damage through its full thickness should be excised (evidence of irreparable damage to deeper layers of skin may not be apparent for several days, and excision in such cases should be done as a secondary procedure). The resulting wound should be handled like any other open surgical wound, primary grafting of skin being carried out, if conditions permit. For painful surgical procedures or dressings, general anesthesia, preferably obtained by intravenous injection, should be employed.

(*c*) Burns of all surfaces except the hands, face, and genitalia are to be treated with tannic acid and silver nitrate. A freshly prepared 10 percent aqueous solution of tannic acid is sprayed over the burned area. This is followed immediately by spraying the area with a mixture of equal parts of 10 percent tannic acid and 10 percent silver nitrate solutions. This mixture should

then be sprayed on the burn every half hour for a total of four applications. If a satisfactory eschar has not been formed, spraying of the mixture should be continued until the objective is attained. Care should be taken to avoid normal skin about the wound. While drying, the burned area may be kept exposed to the air in a heated cradle. After the eschar is dry, it may be covered by a dry sterile dressing.

(*d*) In the absence of infection, the eschar should be allowed to separate spontaneously. If infection develops, the eschar must be removed from the entire infected area, and the latter should then be treated like any other infected wound, with the employment of appropriate systemic and local therapy (par. 51).

(*e*) Tanning has been found to be undesirable in burns of the hands, face, and genitalia. After thorough surgical cleansing, burns of these areas should be covered with a generous application of an aqueous emulsion containing 5 percent of sulfadiazine. If this is not available, boric acid ointment may be substituted. The burned area should then be covered with a fine mesh gauze (44-mesh gauze bandage is satisfactory). A pressure bandage should be applied if the hands are involved in the burn. The dressing should not be disturbed for 10 days unless complications develop.

(*f*) Physiological saline solution has been found useful in the treatment of burns involving the face, hands, and especially fingers, the flexures, and the perineum. It may also be used for the removal of tannic acid or other coagula previously applied and for the treatment of sepsis. Saline may be employed in the form of packs or baths.

(*g*) If for any reason as long as 2 hours must elapse before tanning of a burn can be started, first-aid treatment should be instituted. For burns of all surfaces except the hands, face, and genitalia, this should consist of liberal application to the burned surfaces of a water soluble jelly containing 10 percent of tannic acid and 5 percent of sulfadiazine. If this is not available, boric acid ointment may be substituted. The area is then covered with a sterile dressing. First-aid treatment of burns of the hands, face, and genitalia should consist of a liberal application of a water soluble emulsion containing 5 percent of sulfadiazine. Boric acid ointment may be substituted if necessary. The burn should then be covered with a fine mesh gauze. A pressure bandage should be applied to burns of the hands.

SECTION III

MEDICAL EMERGENCIES

18. General.—As with the preceding section on surgical emergencies, the following paragraphs cover suggestions for the treatment of certain medical conditions that demand more or less immediate action.

19. Acute poisonings.—*a. Acids.*—(1) Neutralize with an alkali, such as magnesia, chalk, white wall plaster, sodium bicarbonate (cooking soda), or lime water.

(2) Give a demulcent, such as milk, olive oil, or egg white.

(3) Keep the patient warm.

(4) Give 0.016 gram (¼ grain) morphine sulfate for pain.

(5) Do *not* pass a stomach tube or give an emetic, because of the danger of perforation.

b. Alcohol.—(1) Acute alcoholism may be confused with a number of other conditions, notably spontaneous or insulin-induced hypoglycemia, cerebral concussion, and acute poisonings due to drugs, particularly cocaine (with acute cocaine poisoning, treatment for acute alcoholism frequently results fatally).

(2) If the diagnosis of acute alcoholism is established, no specific medical treatment is required in most cases.

(*a*) Put the man to bed, cover him warmly enough to prevent chilling, and allow him to sleep until he recovers.

(*b*) The routine use of gastric lavage is not advisable.

(*c*) The patient should be watched until it is certain that the intoxication is not serious.

(*d*) On his awakening, give 1.0 gram (15 grains) sodium bicarbonate and a saline laxative by mouth, if they can be retained; give 0.3 gram (5 grains) acetylsalicylic acid (aspirin) by mouth for relief of general distress; if vomiting persists, give nothing but crushed ice by mouth and administer 0.0006 gram (1/100 grain) atropine sulfate hypodermically, repeating once or twice at 3-hour intervals, if necessary.

(3) If the man is noisy, violent, or combative, and if he is robust—

(*a*) Administer 0.006 gram (1/10 grain) apomorphine hydrochloride hypodermically; this will nearly always quiet the patient and will usually induce vomiting (apomorphine should *never* be given to a drowsy or unconscious patient).

(*b*) If vomiting does not occur, a stomach tube should be passed, and the stomach washed out with several quarts of warm water or of a 5 percent solution of sodium bicarbonate.

(*c*) Barbiturates and other hypnotics should be avoided as far as possible, and should never be given until it is certain that a serious degree of intoxication is not present; paraldehyde is the safest, and may be given in doses up to 8 cc (2 drams), best given orally in grapefruit juice with crushed ice or rectally mixed with olive oil.

(*d*) In rare cases in which apomorphine is ineffective or inadvisable and hypodermic administration of a sedative is deemed necessary, inject 0.2 to 0.3 gram (3 to 5 grains) sodium luminal or sodium amytal; these drugs are too dangerous for general use, since their depressing effect is added to that of the alcohol, and since they occasionally excite rather than quiet the patient.

(4) If a very large amount of alcohol has been imbibed during a short interval, a serious degree of intoxication may develop, which can prove fatal from respiratory and circulatory failure unless energetically treated; such a condition is indicated by drowsiness increasing rapidly to coma; dilated pupils; slow, stertorous breathing; cyanosis; rapid, feeble pulse; loss of reflexes; incontinence; and rarely, convulsions.

(*a*) Insert a stomach tube, taking extreme care that it does not enter the trachea, and wash out the stomach thoroughly with warm water or a 5 percent solution of sodium bicarbonate;

introduce a teaspoonful of sodium bicarbonate and 30 grams (1 ounce) magnesium sulfate, dissolved in a glassful of water, before withdrawing the tube.

(*b*) Take great care that the patient does not get chilled, by keeping him well covered with blankets and by using hot-water bottles, if necessary.

(*c*) The patient should be carefully watched to prevent the aspiration of vomitus and the attendant danger of pneumonia.

(*d*) Inject intravenously a solution containing 25 grams of glucose dissolved in from 50 to 200 cc of water, together with 15 units of ordinary insulin (do *not* give insulin without glucose); this accelerates the rate of oxidation of alcohol in the body.

(*e*) Make the patient breathe a mixture of 10 percent carbon dioxide and 90 percent oxygen to stimulate respiration and hence to increase the rate of excretion of alcohol; this is particularly important if the respirations are depressed.

(*f*) If intoxication is profound, if the respirations are depressed, and if the reflexes are abolished, give metrazol intravenously, not exceeding a dose of 0.1 to 0.3 gram (1½ to 5 grains). Give this slowly, taking a minute to complete the injection; watch the patient carefully and stop the injection if the reflexes return or if twitching of the facial muscles appears; it is not necessary to restore consciousness.

(*g*) If the condition is somewhat less serious, give 0.1 gram (1½ grains) of metrazol hypodermically and repeat once or twice, if necessary; instead of metrazol, 0.3 to 0.5 gram (5 to 7½ grains) caffeine sodium benzoate or 0.25 to 0.50 gram (4 to 7½ grains) nikethamide (coramine) may be given subcutaneously or intravenously.

(*h*) *These last two measures are to be reserved for patients who are seriously ill and are not to be used merely to restore consciousness.*

(*i*) In certain cases, generalized convulsions occur; if mild, they can be controlled by 0.1 to 0.2 gram (1½ to 3 grains) phenobarbital by mouth, or if severe by 0.5 gram (7½ grains) sodium amytal intravenously.

c. *Alkalies.*—(1) Neutralize with a weak acid, such as lemon juice or vinegar.

(2) Keep the patient warm.

(3) Do *not* pass a stomach tube or give an emetic, because of the danger of perforation.

d. Barbiturates.—The following treatment, subject to the needs of the case and limitations of the surroundings, may be carried out:

(1) Pass a stomach tube and wash out the stomach.

(2) Purge with 30 grams (1 ounce) sodium dibasic phosphate.

(3) Inject 0.003 to 0.006 gram (1/20 to 1/10 grain) picrotoxin intravenously, repeating as needed. Strychnine hypodermically 0.002 gram (1/30 grain) to 0.006 gram (1/10 grain) may be used.

(4) Keep the patient warm.

(5) Give continuous oxygen therapy, in combination with carbon dioxide, to stimulate the respiratory center, if necessary.

(6) Aspirate mucus from the trachea, performing endotracheal intubation, if necessary.

(7) Inject 25 to 50 cc of a 50 percent solution of glucose intravenously, if necessary, to promote diuresis and dehydration of the brain and lungs.

(8) Place an indwelling catheter, if necessary.

(9) Perform a lumbar puncture, if necessary, to relieve increased intracranial pressure.

e. Carbon monoxide.—(1) Treat *at once* and continuously.

(2) Supply fresh air and keep the patient quiet.

(3) Apply artificial respiration (par. 29), if necessary.

(4) Provide inhalation of 10 percent carbon dioxide and 90 percent oxygen, if possible.

(5) Relieve cerebral edema by the intravenous injection of 25 to 50 cc of a 50 percent solution of glucose.

f. Cocaine.—(1) Acute cocaine poisoning may follow extensive cocainization or the use of a small amount of the drug in a patient who has an idiosyncrasy to it.

(2) In any operation involving extensive cocainization, give 0.5 to 1.0 gram (7½ to 15 grains) barbital sodium or the equivalent amount of a similar barbiturate by mouth a half hour before.

(3) If signs of overdosage (mental excitement or delirium, with motor restlessness, incoordination, and muscle cramping or convulsions, followed by circulatory and respiratory depression or collapse) appear:

(*a*) Inject *immediately* 0.2 to 0.5 gram (3 to 7½ grains) sodium amytal or sodium luminal intravenously.

(*b*) Repeat, if necessary, using *extreme caution.*

(*c*) If respiratory depression is severe, provide inhalation of 10 percent carbon dioxide and 90 percent oxygen, if possible.

(*d*) If necessary, apply artificial respiration (par. 29).

g. Cyanides (sodium cyanide, prussic acid).—(1) Cyanides act very rapidly, hence *use great haste.*

(2) Inject intravenously 0.3 gram (5 grains) sodium nitrite dissolved in 10 cc water, taking 3 or 4 minutes for injection.

(3) Inject intravenously 25 grams (6 drams) sodium thiosulfate dissolved in 50 cc water, taking 10 minutes for the injection.

(4) Inject 1.0 cc of 1:1000 solution of epinephrine (adrenalin), if the blood pressure falls appreciably.

(5) Pass a stomach tube and wash out the stomach with a 1.5 percent solution of hydrogen peroxide.

(6) Apply artificial respiration (par. 29), if necessary.

(7) Repeat the injections of sodium nitrite and sodium thiosulfate, if necessary.

(8) If sodium nitrite and sodium thiosulfate are not available, inject intravenously 50 cc of a 1 percent solution of methylene blue.

h. Fluorides (chiefly sodium fluoride, in rat poisons and insecticides; also in the aluminum industry).—Ingestion of fluorides is immediately followed by symptoms of acute gastrointestinal irritation—vomiting (often hematemesis), abdominal pains, and diarrhea. Subsequently, attacks of convulsions, spasms, or paresis occur, between which there is generalized weakness. If large amounts have been ingested, death usually takes place in 6 to 10 hours. If a diagnosis of acute poisoning is made—

(1) Pass a stomach tube *immediately* and wash out the stomach with lime water or a 3 percent solution of calcium chloride.

(2) Then give 5 drams (75 grains) calcium gluconate orally or inject 20 cc of a 10 percent solution of calcium gluconate intravenously.

(3) If symptoms of respiratory paralysis develop, inject respiratory stimulants or apply artificial respiration (par. 29).

i. Iodine.—(1) Give a starchy preparation, such as a heaping tablespoonful of cornstarch or flour stirred into a pint of boiling water and the mixture allowed to cool, at once.

(2) Remove the mixture with a stomach tube.

(3) Repeat these two procedures until the solution is no longer colored.

(4) Give 500 to 1,500 cc physiologic saline solution intravenously.

(5) Give morphine sulfate in adequate amounts to relieve pain.

j. Marijuana.—(1) The problem of poisoning from marijuana (*Cannabis sativa*) is rarely concerned with the treatment of acute cases, but rather with the relief of addicted individuals who take the substance either by smoking the leaves or, rarely, by oral consumption of an infusion or alcoholic extract of cannabis.

(2) The mode of action of cannabis is poorly understood and very variable; it usually acts as a descending depressant to the central nervous system, producing in some respects a state resembling alcoholism, with, however, a much greater tendency toward disorientation and dissociation of personality and with a great lengthening of the sense of time.

(3) Death from the taking of even large quantities is rare.

(4) No specific remedy is recommended in the treatment of acute poisoning, other than a thorough gastric lavage, followed by a laxative.

k. Mercury salts (chiefly mercury bichloride).—(1) Give large quantities of egg-white mixed with milk or water.

(2) Then pass a stomach tube and thoroughly wash out the stomach.

(3) Inject 500 to 1,000 cc physiologic saline solution intravenously *at once.*

(4) Give a transfusion of blood or plasma if there is evidence of shock.

(5) If signs of shock develop and a transfusion cannot be performed, give an intravenous injection of 500 cc of a 10 percent solution of glucose and a subcutaneous injection of physiologic saline solution simultaneously, as a temporary expedient.

(6) Give subsequent saline and glucose injections and transfusions in quantities and at intervals so regulated as to ensure in the body the presence of a large, but not excessive, supply of fluid of nearly normal composition.

(7) Give *no* fluids by mouth so long as there is nausea and vomiting.

(8) Diarrhea may be relieved by small cleansing enemas given once a day.

l. Opium (morphine).—(1) Pass a stomach tube and wash out the stomach with a 0.05 percent solution of potassium permanganate.

(2) Inject intravenously 0.5 gram (7½ grains) caffeine sodium benzoate or theophylline.

(3) If stupor has not occurred, make every effort to keep the patient awake and moving.

(4) Inhalation of 10 percent carbon dioxide and 90 percent oxygen may stimulate respiration.

(5) Apply artificial respiration (see par. 29), if necessary.

(6) Give a saline purge—30 grams (1 ounce) magnesium sulfate.

(7) If shock develops, treat as in shock due to other causes (par. 15*c* (5)).

m. Phenol (carbolic acid).—(1) If taken by mouth, pass a stomach tube and wash out the stomach with olive or other edible oils, or if oils are not available use ordinary tap water.

(2) If stomach tube is not available, inject 0.006 gram (1/10 grain) apomorphine hydrochloride.

(3) Inject intravenously 1,000 cc of 5 percent glucose in physiologic saline solution.

(4) Provide inhalation of 10 percent carbon dioxide and 90 percent oxygen or apply artificial respiration (par. 29), if necessary.

(5) If there is a surface burn, wash off the skin with 95 percent alcohol.

n. Phosphorus (in rat paste and matches).—(1) Pass a stomach tube and wash out the stomach with 500 cc of a 0.5 percent solution of copper sulfate.

(2) Repeat at 15 minute intervals.

(3) Give a saline purge—30 grams (1 ounce) magnesium sulfate.

(4) The ingestion of *organic oils* should be avoided.

(5) Give 0.016 gram (¼ grain) morphine sulfate for pain.

(6) Eventually give a demulcent preparation, such as boiled starch solution, eggs, or milk, by mouth.

o. Silver salts (chiefly silver nitrate).—(1) If swallowed, give orally 30 grams (1 ounce) sodium chloride dissolved in a glassful of water; pass a stomach tube and wash out the stomach.

(2) If in the eye, irrigate freely with physiologic saline solution.

p. Strychnine.—(1) Inject intravenously *immediately* 0.5 gram (7½ grains) sodium amytal dissolved in 10 cc sterile water; *inject slowly.*

(2) Repeat, as required, until the patient is in a quiet sleep.

(3) If no amytal is available, substitute another barbital derivative, such as sodium phenobarbital.

(4) Pass a stomach tube and wash out the stomach with a 0.1 percent solution of potassium permanganate.

q. Sulfonamides.—See paragraphs 52 and 53.

20. Bites and stings.—*a. Scabies.*—Apply 5 percent flowers of sulfur in lanolin.

b. Chigger (red mite) *bites.*—(1) For prevention, dust the underclothing and socks with flowers of sulfur or pyrethrum powder (the latter should *not* be used for persons who are allergic to pyrethrum).

(2) Treat bites with phenolated (1 percent phenol) camphor solution in petrolatum to lessen itching and prevent pyogenic infection.

c. Tick bites.—(1) In regions infested with ticks, especially those carrying the rickettsia of Rocky Mountain spotted fever, all clothing should be removed and the body and clothing carefully inspected for ticks at least once a day and preferably every 8 hours.

(2) Attached ticks should be removed without leaving the head in the skin, if necessary by applying a few drops of chloroform or ether.

(3) The ticks should *not* be crushed with the fingers, since both blood and feces may transmit skin-invading rickettsia.

d. Spider bites.—(1) Tarantulas of the Western Hemisphere are said to cause no serious toxic symptoms by their bite, but a lymphangitis and septicemia from secondary infection may occur.

(*a*) Treat the local lesion with wet dressings and, if necessary, surgical incision.

(*b*) Give appropriate chemotherapy (par. 43), if septicemia develops.

(2) Bites of the black widow spider (*Latrodectus mactans*) are quite poisonous.

(*a*) If the patient is seen within a few minutes and if a suction pump is available, apply a tourniquet above the site, paint the bite with tincture of iodine, incise, and attempt to suck out the venom.

(*b*) If absorption of the poison has occurred—

1. Inject intravenously 20 cc of a 10 percent solution of calcium gluconate or intramuscularly the specific antivenom, or both.
2. Give frequent hot baths.
3. Provide adequate sedation.

e. Leeches.—(1) Bites should be painted with tincture of iodine.

(2) After marching through leech-infested water, boots and socks should be removed and examined for leeches; if necessary, the leeches may be made to drop off by applying a strong saline solution.

f. Bee, wasp, hornet, and ant stings.—(1) In nonhypersensitive patients press out the sting and, if convenient, apply a compress of ammonia water or baking soda.

(2) If there are allergic manifestations, inject 0.5 or 1.0 cc of a 1:1000 solution of epinephrine (adrenalin).

g. Snake bites.—(1) Immediately attempt to prevent the venom from entering the circulation by applying a tourniquet above the site of the bite (if possible, apply additional tourniquets at higher levels); the tourniquet should block venous and lymphatic flow but not completely obliterate arterial flow, and should be released for 5 or 10 seconds every 10 or 15 minutes.

(2) As soon as possible, make two or three incisions, 1½ to 2 inches long, through the skin, subcutaneous tissues, and muscle fascia, parallel to the long axis of the extremity, and through fang punctures; if possible, institute suction over these incisions.

(3) Keep the patient quiet.

(4) Give the usual treatment for shock. Give plenty of hot fluids, but *no* alcohol.

(5) If sedatives are needed, use barbiturates, but *no* morphine.

(6) Give cardiac stimulants, if necessary.

(7) Inject 0.006 gram (1/10 grain) strychnine sulfate intramuscularly for respiratory embarrassment, if necessary.

(8) The intravenous injection of 0.5 gram (7½ grains) calcium chloride may lessen hemolysis.

Caution: If used, this should be injected *slowly* and care should be employed that none is injected outside the vein.

(9) In all seriously poisoned cases, the patients should be treated for shock and hospitalized as soon as possible.

(10) The intravenous injection of 500 to 1,000 cc of a 5 or 10 percent solution of glucose is helpful if hemolysis occurs, but repeated blood transfusions are required if the hemolysis is marked.

(11) Specific antivenoms are available for the treatment of bites of certain snakes; if the proper antivenom is available, the directions accompanying the package should be followed, taking the usual precautions against sensitivity to horse serum (par. 22).

(12) Even if antivenom is administered, blood transfusions are usually necessary.

(13) Precautions should be taken against tetanus infection (par. 49).

h. Jellyfish, Portuguese man-of-war, and nettle stings.—(1) Dry the skin promptly, and apply pressure to relieve pain.

(2) Scrub the skin with soap and water to remove the remnants of tentacles, and apply an alcohol dressing.

(3) When dermatitis develops, accompanied by itching, apply a calamine lotion containing 1 percent phenol.

(4) For bullae, use wet, boric acid compresses.

(5) For dry, itching lesions, apply carbolized vaseline.

(6) If constitutional symptoms (muscle cramps, respiratory embarrassment, and collapse) develop, appropriate symptomatic therapy should be given.

i. Animal bites (rabies).—(1) Since rabies may be contracted from the bite of any rabid animal, the animal, usually a dog, unless known to be rabid, should be incarcerated, if possible, and watched for at least 10 or 15 days to determine whether the symptoms and signs of rabies develop.

(2) Immediately open the wound freely, cleanse it, and then thoroughly cauterize all parts of the exposed tissues with pure carbolic acid, after which 95 percent alcohol should be applied (nitric acid is as efficacious as carbolic acid, but is more painful).

(3) If the animal is known to be or suspected of being rabid or if the bite is on the head or face, start Pasteur treatment immediately, and continue the inoculations until it is proved whether or not the animal has rabies.

(4) If the bite is on the extremities or body, treatment may be postponed until the animal is proved to be rabid.

(5) If doubt arises as to the diagnosis, Pasteur treatment should be started promptly.

21. Poison ivy (*Rhus radicans*) and poison oak (*Rhus diversiloba*) dermatitis.—*a. Treatment.*—(1) Bathe the exposed parts with a strong soap solution, followed by alcohol; after the dermatitis has developed, the use of soap and water should be minimized.

(2) In the erythematous stage, use a calamine lotion containing 1 percent phenol or menthol.

(3) In the exudative stage, apply dressings soaked with 4 percent boric acid, 10 percent aluminum acetate, or 0.02 percent potassium permanganate solution, using cold rather than warm applications.

(4) In the exfoliative stage, apply boric acid ointment or carbolated vaseline.

(5) At no time should strong medications be used.

b. Prevention.—The following protective procedure is recommended by the National Institute of Health [1] for the prevention of poison ivy dermatitis:

(1) An alkaline vanishing cream [2] containing a nonirritant oxidizing agent, such as sodium perborate or potassium periodate.

(2) It should be well rubbed into the skin of the arms and face of persons before exposure to poison ivy. This procedure leaves a deposit of the powdered oxidant on the skin.

(3) The protective cream should be allowed to remain on until the noon hour when it should be removed by washing with soap and water; this will emulsify the vanishing cream in the pores of the skin and wash away from within outward whatever toxin may be in the pores or on the skin.

(4) The cream should be reapplied again after the lunch hour and again washed off in the evening when work is over.

(5) This vanishing cream should be freshly prepared at least once in 2 weeks to avoid deterioration.

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[1] Schwartz, Warren, Goldman—Public Health Reports, Vol. 55, No. 30, Page 1327, July 26, 1940.

[2] Stearic acid (triple pressed) 200 gm, potassium hydroxide (sticks) 14 gm, water 800 cc, alcohol (90 percent) 40 cc. To 50 gm of this cream, 5 gm of sodium perborate is added.

22. Allergic and anaphylactic reactions.—*a. Allergic asthma.*—(1) This type of asthma should be differentiated from that due to cardiac disease, edema of the larynx, massive lesions in the mediastinum, infection of lungs, and gassing.

(2) Treat as follows:

(*a*) Give 0.3 to 0.5 cc of a 1:1000 solution of epinephrine (adrenalin).

(*b*) This may be repeated at 20-minute intervals.

(*c*) For a prolonged effect, inject 1.0 cc of a 1:500 solution of epinephrine (adrenalin) in oil intramuscularly.

b. Edema of larynx (angioneurotic edema).—(1) Inject 0.5 cc of a 1:1000 solution of epinephrine (adrenalin).

(2) Repeat two or three times at 15-minute intervals, if necessary.

(3) If tracheotomy is indicated, do not delay until deep cyanosis occurs or until the patient is exhausted.

c. Serum reactions.—Several types of reaction may follow the parenteral injection of foreign serum.[3]

(1) Collapse or syncope may occur after any hypodermic medication.

(*a*) Treat as for syncope in general.

(*b*) If the blood pressure becomes dangerously low, inject 0.5 cc of a 1:1000 solution of epinephrine (adrenalin).

(2) Atopic or anaphylactic reactions follow the injection of serum into susceptible persons. Itching, hives, asthma, and vomiting may be followed promptly by collapse or even death. These phenomena occur immediately in a naturally sensitive person or after 1 to 5 hours in those sensitized by the previous injection of serum.

(*a*) Previous to the injection of *any* foreign serum, the following precautions should always be observed:

1. Question the patient concerning a personal or family history of asthma, particularly so-called horse asthma, hay fever, or other hypersensitivity; serum should *not* be given to a patient with a positive history of asthma, unless in the opinion of the medi-

[3] The term "serum" means prophylactic or therapeutic serum obtained from the blood of animals, and not vaccines, pollen extracts, and other materials, which are at times loosely and improperly called serums. In general, instructions included with the commercial packages of therapeutic and prophylactic serums should be followed.

cal officer it is vitally necessary and then only with greatest caution (par. 44).

2. Perform intradermal and ophthalmic tests for sensitivity (par. 44); serum should *never* be given if the ophthalmic or both reactions are positive unless the patient can be desensitized, and *with the greatest caution* if the skin reaction is positive.

3. Always have on hand a sterile syringe filled with a 1:1000 solution of epinephrine (adrenalin).

(*b*) When a reaction does occur—

1. If the injection was given subcutaneously or intramuscularly, apply a tourniquet, if possible, above the site of injection, leaving it in place for 20 or 30 minutes and releasing it for a few seconds every few minutes.

2. Inject *immediately* 1.0 cc of a 1:1000 solution of epinephrine (adrenalin) intramuscularly; if a tourniquet has been applied, injection should be made proximal to it.

3. Repeat this dose every 5 to 15 minutes, as needed.

4. Apply general measures, such as bedrest and warmth, for the treatment of collapse.

(3) Serum sickness cannot be prevented; it begins 5 to 7 days after the injection and lasts for 3 or 4 days, being characterized by urticaria, fever, lymph node enlargement, arthralgia, alone or in combination. Inject, as needed, 0.5 cc of a 1:1000 solution of epinephrine (adrenalin) for hives, and give 0.3 to 1.0 gram (5 to 15 grains) acetylsalicylic acid (aspirin) for joint pains and fever.

d. Transfusion reactions.—If substernal oppression, anxiety, back pain, or flushing of face develop—

(1) Stop the transfusion.

(2) Inject *immediately* 0.5 to 1.0 cc of a 1:1000 solution of epinephrine (adrenalin) subcutaneously or intramuscularly.

(3) Administer sodium bicarbonate orally in sufficient amounts (5 grams (75 grains) every 4 hours) to render the urine alkaline, and continue to keep the urine alkaline for 48 hours.

(4) Certain allergic manifestations may require additional injections of epinephrine (adrenalin).

(5) Anuria, oliguria, or hematuria is an indication for *immediate* hospitalization.

23. Food intoxications and poisonings.—*a. Food intoxications.*—Acute simple diarrhea is usually due to irritating substances present in spoiled or contaminated (with certain paratyphoid bacilli or staphylococci) food, to poor kitchen sanitation, or to improper care of eating utensils. (The more serious types of diarrhea, dysentery, typhoid and paratyphoid fever, and cholera are due to specific infections.) Rigid supervision of the storage, preparation, and distribution of food, and strict adherence to regulations relative to the care of mess kits greatly reduce the incidents of diarrheal disorders. It is imperative that regulations relative to these matters be adhered to if epidemics are to be avoided in encampments and in the field. If acute simple diarrhea occurs—

(1) Within 12 hours, if there is no nausea, give 30 cc (1 ounce) castor oil.

(2) If heat to the abdomen does not control pain give 8 cc (2 drams) paregoric or 0.06 gram (1 grain) codein orally; in severe cases, morphine may be required.

(3) Limit food to a liquid or bland diet, even for several days after recovery.

(4) When the stools are watery and contain but little fecal material, give 4 cc (1 dram) paregoric in warm water with 1 gram (15 grains) bismuth subcarbonate every hour until the diarrhea is controlled.

(5) If shock is marked, the usual treatment should be given (par. 15*c*(5)).

(6) Unless improvement promptly occurs, hospitalization is indicated.

(7) Protect others from possible infection from vomitus or feces.

b. Food poisonings.—(1) *Botulism.*—The appearance, usually within 48 hours after ingestion of questionable food, of central nervous system symptoms, such as dizziness, disturbances of vision, speech and swallowing, weakness and incoordination, without fever, is suggestive of botulism, rarely of mussel poisoning. The possibility of encephalitis or poliomyelitis should be considered in differential diagnosis. If a diagnosis of botulism is made—

(*a*) Inject *immediately* 50,000 units bivalent antitoxin, diluted with 1,000 cc of a 5 percent solution of glucose, intravenously, taking the usual precautions in administering horse serum (pars. 22 and 44).

(*b*) In the very early stages, gastric and colonic lavage may be employed.

(*c*) Hospitalize *at once*, since these patients bear moving badly after the first 12 hours.

(*d*) Avoid food and drink by mouth, since there is danger of *strang*ulation from pharyngeal paralysis.

(*e*) Asymptomatic partakers of the suspected food should be given 2,500 units bivalent antitoxin intramuscularly.

(2) *Mussel poisoning* (shellfish paralysis).—This is due to the ingestion of any shellfish, usually mussels, that contains quantities of plankton organisms. This occurs oftenest on the Pacific coast during the months from June to September, inclusive, when plankton are present in large numbers in the sea water, as is evidenced by phosphorescence. Prevention is possible by the avoidance of the eating of any mussels or clams during these months. Cooking does not destroy the poison. Death, if it occurs, comes within the first 12 hours, usually from paralysis of the respiratory center. The ataxia and other symptoms usually disappear in nonfatal cases. If the diagnosis is made—

(*a*) Increase elimination.

(*b*) Apply artificial respiration (par. 29), if necessary.

(3) *Mushroom poisoning.*—The symptoms may be immediate or delayed (48 hours). Vomiting, abdominal cramps, severe diarrhea, fixed contracted pupils, and mental confusion occur, and in certain cases, jaundice, anemia and hemoglobinuria. In cases with delayed poisoning, usually due to *Amanita phalloides*, treatment, other than supportive, is of no avail. If the diagnosis of acute poisoning is made—

(*a*) Pass a stomach tube immediately and thoroughly wash out the stomach.

(*b*) Give repeated enemas.

(*c*) Give 30 grams (1 ounce) magnesium sulfate by mouth.

(d) Inject 0.006 gram (1/100 grain) atropine sulfate subcutaneously, and repeat if physiologic effects (dilation of the pupils and dryness of the mouth) are not obtained.

24. Care of feet.—*a. Foot hygiene.*—(1) Healthy function of feet can be maintained only when rigid hygienic precautions are continually enforced by frequent, periodic inspection.

(2) Bad foot hygiene results from failure to keep the feet clean and from trauma to the feet.

(3) Proper cleanliness necessitates thorough washing with soap and water, particularly between the toes, and scrubbing of the nails, followed by complete rinsing. The nails should be trimmed transversely and in such a manner that the lateral edges are never cut back toward the cuticular attachment.

(4) Excessive moisture plays an important role in maceration of the skin and proliferation of surface bacteria and fungi. Socks should be changed as often as is practicable, especially during forced marches. Persons whose feet perspire excessively should change socks and shoes at midday whenever possible, and should never wear oiled shoes. The following dusting powder is of value: the Army foot powder composed of salicylic acid 3 percent, boric acid 5 percent, starch 10 percent, talcum powder 82 percent.

(5) Trauma to the feet originates from poorly fitting shoes or from the application of force in excess of that to which the feet have been accustomed, and usually follows forced marches. Soldiers' shoes should be fitted to avoid points of constriction or pinching; the shoe pressure should be uniformly distributed. They should be laced snugly and carefully inspected with the wearer standing erect and bearing a weight equivalent to that of a soldier's pack. New shoes should never be worn on march. Socks should be fitted to the size of the foot. In general, because of their greater absorbability, light wool issue socks are preferable.

(6) The gradual accommodation of the feet to increasing weight bearing, together with cleanliness and attention to footwear, minimizes the incidence of foot injuries. The following are suggestions relative to the care of the feet after long marches:

(*a*) Wash the feet in cold water; after drying, use a dusting powder.

(*b*) If the skin is tender, bathe the feet in warm salt water or alum water; if the feet are swollen and perspiring, soak them in a 2 percent solution of formalin; if they are very tender soak them in a 1 percent solution of formalin or a 10 percent solution of picric acid.

(*c*) Open blisters with a sterile needle, and circle them with adhesive tape to lessen pressure.

(*d*) Dry and stretch the shoes.

(*e*) Wash and dry the socks.

(7) The importance of a foot bath with soap and water cleansing, vigorous massage (20 minutes), dry socks, and a change of shoes in the prophylaxis of foot disability resulting from exposure to cold and moisture cannot be overemphasized.

b. Trichophytosis.—(1) Proper cleansing and the control of moisture greatly reduce the incidence of so-called "athlete's foot." Prevention of infection in company bathhouses may be attempted by requiring a foot bath of a few minutes in a solution of calcium hypochloride or sodium hypochloride giving 50–100 parts per millim of available chlorine. An effective remedy is the use of the Army foot powder consisting of salicylic acid 3 percent, boric acid 5 percent, starch 10 percent, talcum powder 82 percent. The powder is rubbed on the feet and used as a dusting powder for the feet and socks.

(2) Those with simple scaling between the toes and occasional blisters are rarely disabled except during forced marches or when the feet have remained wet for prolonged periods; their feet usually respond promptly to rubs with foot powder two or three times daily.

(3) When inflammation is acute, wet packs of a 4 percent solution of boric acid or aluminum acetate are useful.

(4) In long-standing infections associated with hyperkeratosis, preliminary treatment (four to seven nightly applications) with either 5 percent salicylic ointment or Whitfield's ointment, followed by the Army foot powder is effective.

(5) If the foregoing measures fail, the feet may be painted daily for 4 days with half-strength (3½ percent) tincture of iodine, then once weekly for 4 weeks; the iodine solution should be applied to the entire foot, ankle, and the free edges of the nails, and promptly removed with 60 percent alcohol.

(6) Fumigation of infected shoes may be accomplished by inserting in each of them a piece of blotting paper containing a teaspoonful of formalin and wrapping them tightly in paper for 24 hours. They should be aired thoroughly before using.

(7) When the lesions become secondarily infected, with resulting cellulitis and lymphangitis, the constant application of dressings saturated with hot solutions of 4 percent boric acid or physiologic saline solution are indicated, together with appropriate specific chemotherapeutic agents (par. 43).

25. Seasickness.—*a.* The early symptoms and signs of seasickness are a sense of tightness of the throat, salivation, anorexia, and frequent swallowing. If the stimuli of motion

are prolonged, the patient becomes pale and nauseated, and clammy skin, vertigo, incoordination of the muscles, and mental lethargy become prominent symptoms. In very severe cases cyanosis, thirst, subnormal temperature, tachycardia, and a drop in blood pressure are the outstanding features. If vomiting continues over a long period of time, the urine becomes scanty and highly colored, acetone may be present, and the resulting dehydration may produce a polycythemia with red cell counts of 7,000,000 to 8,000,000. The disorder is never fatal in an otherwise normal person.

b. The treatment of seasickness is notoriously unsatisfactory.

(1) Prophylactic measures of various types have been recommended;

(*a*) Plugging the ears may prevent seasickness.

(*b*) In obese persons and in those with relaxed abdominal muscles, the wearing of an abdominal support has proved useful in the prevention of plane sickness, and in the prevention of true seasickness in very susceptible individuals.

(*c*) Moderate physical activity in the open air and mental diversion should be advised, as well as rhythmic breathing, with inspiration timed with the rise of the boat and expiration with its fall.

(*d*) A small amount of food and liquid should be taken at frequent intervals, and alcoholic liquors should be limited to very moderate quantities. Bromides, phenobarbital, and chloral hydrate may be given by mouth in small doses.

(2) If the patient is subjected to seasickness for a prolonged period, severe dehydration may require the injection of fluids intravenously, preferably 10 percent glucose in physiologic saline solution.

26. Altitude sickness.—*a.* Altitude sickness, or mountain sickness, results from a diminished partial pressure of oxygen in the environmental atmosphere. An altitude of 25,000 feet is the utmost limit to which ascent may be safely made without the use of oxygen. The usual symptoms—headache, vertigo, physical and mental inertia, dyspnea, palpitation, nausea, vomiting, impaired hearing, and psychic disorder—occur with a fair degree of consistency in the unacclimatized person subjected to altitude greater than 12,000 feet; but the greater the altitude and the more rapid the ascent, the more severe and varied the symptoms.

b. Oxygen inhalation should be begun at altitudes above 10,000 feet, since individual susceptibility varies, and there are variations in the same person from day to day; it promptly relieves the symptoms.

c. Rest is imperative for the relief of the fatigue and mental disturbances.

27. Compressed-air illness.—*a.* Compressed-air illness (caisson disease, the "bends," or divers' palsy) results in persons who, after breathing air under a pressure of considerably greater than 1 atmosphere, are subjected to unduly rapid reduction of air pressure. The symptoms, pain in the back, paraplegia, incoordination, and incontinence of urine, are due to the release in the tissues of bubbles of atmospheric nitrogen that have been dissolved in the body fluids under high atmospheric pressure; these bubbles, when sufficiently profuse, obstruct the circulation.

b. Treatment consists in recompression and subsequent slow decompression; however, there are at present no truly standardized methods for accomplishing this.

c. A recommended procedure is to place the patient immediately after the onset of symptoms in a steel chamber or lock, where the pressure is raised, by admitting pure oxygen, until all symptoms are relieved, 1 additional atmosphere of pressure then being created to effect complete restoration of blood flow to the tissues; this pressure is maintained for a minimum of 30 minutes, and then the pressure is gradually reduced until decompression to normal atmospheric pressure is reached.

28. Electric shock.—*a.* Many factors govern the effects of electric currents on the body. Currents of low voltage arrest the heart without affecting the respiration; alternating currents of low tension induce ventricular fibrillation; high-tension currents cause inhibition of respiration. Death may be due to prolonged muscular tetany resulting in asphyxia, to ventricular fibrillation, to respiratory failure, or to the delayed effects of burns.

b. After the victim is freed from the current, *immediately* apply artificial respiration (par. 29), continuing until natural breathing is restored or until rigor mortis sets in; the administration of stimulating drugs and the injection of epinephrine (adrenalin) are *not* recommended.

29. Immersion.—*a.* In cases of immersion, time is the essential factor; every minute lost after cessation of breathing decreases the chance of recovery.

b. Start artificial respiration immediately, the preferable procedure being the prone-pressure or Schaefer method, which is as follows:

(1) Lay the patient face down, one arm extended directly overhead, the other bent at the elbow with the face turned to one side and resting on the hand or forearm, so that the nose and mouth are free for breathing.

(2) Kneel, straddling the patient's hips, with the knees just below the patient's hip bones or the opening of pants' pocket; place the palms of the hands on the small of the patient's back, with the fingers over the patient's ribs, the little fingers just touching the lowest ribs, the thumbs alongside the fingers and the tips of the fingers just out of sight.

(3) While slowly counting "one," "two," and with the arms held straight, swing forward slowly, so that the weight of the body is gradually, but not violently, brought to bear on the patient; this should take from 2 to 3 seconds.

(4) While counting "three," swing backward to remove the pressure.

(5) While counting "four," "five," rest.

(6) Repeat these operations deliberately, swinging forward and backward twelve to fifteen times a minute, thus making a complete respiratory cycle in 4 or 5 seconds.

(7) As soon as artificial respiration has been started, and while it is being continued, an assistant should loosen all tight clothing about the patient's neck, chest, and waist, and wrap the patient with a blanket.

(8) Continue artificial respiration without interruption until natural breathing is restored or until rigor mortis has set in; do *not* stop merely because the patient appears to be dead.

(9) If natural breathing stops after being restored, use this method of resuscitation again.

30. Heat exhaustion, heat stroke, and heat cramps.—*a.* Exposure to a high environmental temperature, particularly when associated with high humidity, may result in a variety of indefinite subjective symptoms. On the other hand, three definite patterns may appear: heat exhaustion, characterized by muscular weakness, vertigo, profuse sweating, accelerated

pulse, and lowered blood pressure; heat stroke, or sun stroke, characterized by headache, vertigo, nausea, visual disturbances, and loss of consciousness, the temperature being elevated in severe cases to 109° F. or more and the pulse being full and rapid, the face flushed, and the skin dry and hot; and heat cramps, characterized by painful spasms of the voluntary muscles, particularly those of the extremities and abdominal wall.

b. Heat exhaustion usually requires only rest in a cool, shaded room; if the collapse is prolonged, 1,000 cc of physiologic saline solution should be injected intravenously, the head should be lowered by elevating the foot of the bed, and warmth should be applied to the extremities; the intramuscular injection of 0.5 cc 1:1,000 epinephrine (adrenalin) or of 0.5 gram (7½ grains) caffeine sodium benzoate may be necessary.

c. Heat stroke demands prompt treatment. An ice-water tub bath is the most effective measure to reduce body temperature, and the patient should be kept in the bath until the rectal temperature is reduced to 102° F.; if there is a recrudescence after removal to a cool room, the patient should again be placed in the bath.

d. Heat cramps may be prevented by supplying men working in high temperatures with salinized water (a 0.1 percent solution of sodium chloride, cooled to about 50° F.); the cramps are promptly relieved by the intravenous injection of 1,500 cc physiologic saline solution, this quantity being repeated every 4 hours so long as the muscular cramps persist.

31. **Exhaustion.**—*a.* (1) Physical exhaustion implies an exaggeration of the physiologic processes of fatigue; it involves loss of water through increased respiration, combustion, and sweating, salt loss from excessive sweating, and a depletion of the glycogen, fat, and protein stores.

(2) Therapy consists of replacement.

(*a*) Restore the fluid and salt, particularly under conditions of elevated temperature and humidity, by the administration of a 1 percent solution of sodium chloride by mouth or physiologic saline solution intravenously.

(*b*) Give frequent feedings of moderate amounts of food, at first simple sugars and subsequently a balanced diet (daily requirements for violent exercise are ordinarily between 5,000 and 8,000 calories).

(*c*) Rest, aided by oral or parenteral sedation (sodium amytal, phenobarbital, or bromides), is frequently an urgent necessity.

b. Nervous exhaustion may be associated with loss of fluid, salt, and deposit substances, but fundamentally requires sedation; when it is associated with pain due to trauma, morphine may be indicated in preference to sedatives.

c. For heat exhaustion see paragraph 30.

32. Starvation.—The treatment of starvation is that of replacement, orally or parenterally, under resting conditions, of certain fundamental substances, particularly carbohydrates, salt, and water.

a. In acute starvation, the patient should be given warm drinks, fruit juices, honey, gelatine, cooked cereal, and glucose in moderate amounts and sodium chloride in sufficient amount to restore the salt content of the body fluids.

b. If dehydration exists (as evidenced by a dry tongue and oliguria) and swallowing is difficult, slowly administer 1,000 to 2,000 cc of 10 percent glucose in physiological saline solution parenterally, repeating at 6-hour intervals, if necessary.

c. If the starvation has been of long duration, employ frequent feedings of warm liquids for 1 or 2 days and administer accessory food substances, particularly vitamins B_1 and C, orally or parenterally, to prevent the acute appearance of such disorders as scurvy, pellagra, and beriberi.

d. If starvation is complicated by hemorrhage, give plasma or whole blood transfusions.

33. Frostbite.—*a*. Frostbite results from prolonged peripheral vasoconstriction induced by exposure to cold. The local injury varies from a simple erythema to secondary thrombosis, livid cyanosis, or gangrene.

b. Prophylactic measures include the wearing of warm, dry clothes, reasonable physical activity, and the avoidance of alcohol and smoking during and immediately before exposure.

c. If frostbite occurs—

(1) Put the patient in a warm room and give a warm drink, preferably coffee, which may be advantageously combined with spirituous liquors.

(2) The frozen limb should be gently pinched and massaged starting at its junction with the trunk and working distally; rub with a coarse, dry towel, taking care not to injure the skin.

(3) When a joint is passed, it should be subjected to slight passive movement without force, followed by active movement.

(4) Frost blisters should not be disturbed.

(5) Nupercaine one percent ointment may be applied to relieve local discomfort.

34. Sunburn.—*a.* Sunburn is an inflammation of the skin from the actinic energy in sunlight. The intensity of the reaction is in ratio to the length of exposure and the pigment status of the individual, red blondes being the most vulnerable. The inflammation varies from a simple erythema or redness to an acute dermatitis with vesicles and bullae.

b. Sunburn may be prevented by application to the skin of a heavy-bodied vegetable oil such as olive oil or a metallic powder such as zinc stearate or zinc oxide.

c. If simple erythema develops, apply mentholated (0.2 percent) cold cream.

d. If erythema with slight vesiculation is present, a lotion, such as calamine lotion, with or without 1 percent phenol, is preferable.

e. If there is acute dermatitis with the formation of bullae, the patient may require hospitalization. For 24 to 48 hours, apply detergent packs soaked in a 5 percent solution of aluminum acetate or, if the skin is unbroken, a saturated solution of magnesium sulfate. Then use a drying lotion, such as calamine lotion. When the skin begins to crack and peel, apply a salve, such as zinc oxide ointment.

35. Acute psychoses and related conditions.—*a. General.*—(1) Neuropsychiatric disabilities are vital medico-military problems because of their incidence and because their presence endangers the morale and discipline of troops.

(2) The objective treatment is twofold: to return to duty as many men as possible and to minimize the consequence of disability.

b. Diagnosis.—(1) The diagnosis of the underlying condition is rarely obvious. The widest variety of symptoms and signs occurs, but allowing for many combinations, the outstanding symptomatic segments are—

(*a*) Physical reactions, ranging from convulsions, coma, lesser degrees of disturbances of consciousness, paralyses, deafness, blindness, and severe headache, to tics, limited anaesthesias, and other milder symptoms.

(*b*) Emotional reactions, including depression, often suicidal, elation, resentment, irritability, fear, suspicion, excitement, anger, rage, and homicidal tendencies, with various admixtures.

(*c*) Military misbehavior, embracing suspected fifth-column activities or spying, arousing and spreading discontent, inciting insubordination, striking an officer, drunkeness, neglect of duty, cowardice, sulkiness, quarrelsomeness, and petty stealing.

(2) Examination should be extensive enough to answer the following questions:

(*a*) Is there definite evidence of underlying structural lesion or disease?

(*b*) If so, is the seat of the disease outside the nervous system (uremic convulsions, sinus headache, or other organic disease)?

(*c*) Is it psychogenic in origin?

(*d*) If psychogenic, is it psychosis or psychoneurosis?

(*e*) If psychoneurosis, is it the usual war neurosis (conversion hysteria) or a less frequent neurosis (neurasthenia or an anxiety or obsessive-compulsive neurosis)?

(*f*) Can the disabling symptoms be speedily removed?

(*g*) Is it malingering?

(3) Elaborate histories cannot be expected, but reliable sources of information are the sick soldier's officers, his noncommissioned officers, and other soldiers who are his intimates.

(4) A brief neurologic examination, including pupillary responses, tendon reflexes, abdominal and cremasteric reflexes, Romberg, posture, and gait, often furnishes valuable diagnostic leads.

(5) The mental examination is largely dependent on careful observation—general appearance, state of body, clothing, facial expression, attitude, motor activity (purposeful or aimless, related or unrelated to the environment), catalepsy, stupor, mannerism, negativism, suggestibility, echopraxia, and other functional manifestations.

(6) The soldier should be discriminatingly questioned in order to confirm impressions of observational data and to determine the presence of decided mood alternatives, overactivity or underactivity of thought and speech, illusions, hallucinations, obsessions, ideas of reference, delusions, lack of orientation, and disturbances of memory.

(7) An estimate of the emotional state and of consciousness is a necessary condition of diagnosis and treatment. In manic depressive states the emotional display is likely to be fairly clear-cut, depressed, often with self-blame and suicidal trends, or exhiliarated with quick shifts to other emotional reactions, including rage leading to dangerous violence; in schizophrenia, the surface emotional expressions tend to be inadequate to the verbally expressed thinking or even at odds with it.

(8) In the recoverable war neuroses, particularly those occurring in combat, there is often an initial befogged state, perhaps actively precipitated by concussion, fatigue, or food deprivation; even more favorable are acute psychoses with delirium dependent upon physical exhaustion.

(9) Malingering is a deliberately planned attempt to evade military duty or to secure a discharge by feining illness; while not easy to detect, yet usually the simulation is overdone or incomplete with the absence of fundamental signs and symptoms.

(10) Military misbehavior is frequently incipient evidence of psychosis, psychoneurosis, or even organic neurologic disease; if it has a psychogenic basis, successful treatment depends on uncovering the underlying condition and dealing with it.

(11) The common war neurosis is conversion hysteria, in which there is an underlying unconscious conflict—the demands of the instinct of self-preservation, strongly activated by moving emotions, fear, horror, and revulsion versus the ideals of soldierly duty, patriotism, honor, training, and discipline reactions. Under the added burden of conscious worry, stress, deprivation, fatigue, or physical shock-like concussion, the conflict may be converted into protective symptoms.

(12) Conversion hysteria is more likely to occur in the intellectually average soldier than in the superior soldier and officer, and its symptoms appear much more abruptly than in the other neuroses, and are more apt, especially in combat, to be total blindness, paralyses, and hemianesthesias, rather than blurring of vision, weakness, and paresthesias.

c. *Treatment.*—(1) Proper psychiatric treatment should include—

(*a*) Establishment of rapport.

(*b*) An attempt to remove some of the symptoms by persuasion and suggestion, given verbally and sometimes with the aid of some instrumental device like a tuning fork.

(*c*) Desensitization and simple explanatory therapy.

(*d*) The production of positively and negatively conditioned psychic reflexes—positive, concerning the advantages of returning to the front or to the camp; negative, painting evacuation in gloomy colors.

(2) A few general rules are as follows:

(*a*) Give each patient on admission a hot drink.

(*b*) Each patient should have three full meals a day, unless otherwise ordered.

(*c*) Do not discuss symptoms with the patient.

(*d*) Be firm and optimistic in all dealings with the patient.

(*e*) No one should be permitted to see the patient unless assigned for duty.

(3) In the milder states of excitement, 2 to 8 cc (½ to 2 drams) paraldehyde or 0.2 to 0.5 gram (3 to 7½ grains) sodium amytal may be given by mouth.

(4) For depressed states, particularly when associated with slow thinking, give 0.005 to 0.020 gram (1/12 to ⅓ grain) amphetamine (benzedrine) sulfate each morning by mouth.

(5) In more active excited states, inject 1 to 2 cc (15 to 30 minims) paraldehyde or 0.3 to 0.6 gram (5 to 10 grains) sodium amytal diluted with distilled water, intravenously.

(6) Violence, particularly suicidal or homicidal tendencies, must be *immediately* controlled, by mechanical restraint if necessary.

(7) It should always be borne in mind that, in the majority of cases, early treatment increases the chance of cure.

SECTION IV

DIAGNOSIS AND TREATMENT OF VENEREAL DISEASES

36. General.—This section on the diagnosis and treatment of venereal diseases is based on the opinions and recommendations of the Committee on Chemotherapeutic and Other Agents

and its Subcommittee on Venereal Diseases, Division of Medical Sciences, National Research Council, as published in Circular Letter Number 18, Office of The Surgeon General, March 10, 1941. Because of the rapid development of chemotherapeutic agents, it is likely that some of these recommendations will have to be modified from time to time. Certain measures other than chemotherapeutic are described where appropriate. Commonly accepted therapeutic or nursing procedure should not be excluded nor neglected.

37. **Gonorrhea.**—*a. Diagnosis in male.*—(1) A diagnosis of gonorrhea must not be made in the absence of laboratory confirmation, although in emergencies, where laboratory facilities are not available within 24 hours, patients with acute purulent urethral discharge should be started on treatment for gonorrhea.

(2) Acute gonorrhea (anterior urethritis) is diagnosed on the basis of a purulent urethral discharge with gram-stained smear positive for gram-negative intracellular diplococci.

(3) Chronic gonorrhea (posterior urethritis, prostatitis, seminal vesiculitis, or arthritis) is diagnosed on the basis of a positive gram-stained smear of urethral discharge or prostatic secretion or by positive culture of appropriate body fluids.

(4) Gonorrheal ophthalmia (an acute purulent conjunctivitis, rapidly progressing to involve other external coats of the eye) is diagnosed by positive gram-stained smears of the secretion.

b. Diagnosis in female.—(1) Definite diagnosis of early gonorrhea in women is more difficult than in men.

(2) Diagnosis should be based on—

(*a*) *History.*

1. Symptoms (dysuria, vaginal discharge, vulvar pruritus, pelvic inflammation, or acute arthritis).

2. Exposure to known cases of gonorrhea.

(*b*) Clinical examination, especially abdominal, pelvic, and rectal (particularly important in chronic gonorrhea).

(*c*) Gram-stained smears of secretions from the urethra, Bartholin's glands, cervix, and rectum.

Caution: The normal genital flora may contain organisms that closely resemble gonococci.

(*d*) Culture, which should always be performed if smear findings are not corroborated by the history and physical examination.

c. Treatment of acute gonorrhea in male or female.—(1) *Local treatment (male).*—(*a*) Irrigations, instillations, and other local treatment should not be used in any form except in sulfonamide-resistant patients or in those with complications (posterior urethritis, prostatitis, and seminal vesiculitis); and then only by specially trained personnel in a dispensary or hospital.

(*b*) In acute cases, local treatment may do more harm than good.

(2) *Local treatment (female).*—(*a*) In the acute stage, external cleanliness only; *no douches.*

(*b*) In acute pelvic inflammatory disease—

1. Bed rest.

2. Ice bags to abdomen.

3. Keep bowels open; if enemas are necessary, clean perineum carefully before inserting a rectal tube, to avoid infecting the rectum.

4. Hot, cleansing douches.

(3) *Chemotherapy (male and female).*—(*a*) Even under ambulatory conditions, acute gonorrhea may be cured by appropriate measures in a large proportion of cases; therefore, when considered desirable and local conditions permit, acute gonorrhea may be treated on an ambulatory basis. Hospitalization is necessary for the treatment of complications.

(*b*) Sulfathiazole and sulfapyridine are better than sulfanilamide.

(*c*) In the dose advised below, neither sulfathiazole nor sulfapyridine is likely to cause serious toxic manifestations. (See pars. 52 and 53.)

(*d*) If sulfathiazole fails, use sulfapyridine (see below); if neither is available use sulfanilamide.

(*e*) Other medication by mouth (copaiba, sandalwood oil, or alkalies) is unnecessary and should not be used.

(*f*) The scheme of treatment [4] should follow the outline below:

1. Plan A.

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[4] Before treatment, a careful inquiry should be made as to previous sulfonamide therapy, being especially careful to exclude the possibility of self-administered chemotherapy.

Day or days (incl.)	Drug	Daily dosage		Single dose		Time of administration
		Gm	*Gr*	*Gm*	*Gr*	
1st	Sulfathiazole	3.0	45	0.5	7½	Every 3 h., 6 AM to 9 PM
2d to 4th	Sulfathiazole	2.0	30	0.5	7½	Every 5 h., 6 AM to 9 PM
5th	Examine patient; if discharge persists, stop sulfathiazole and switch to plan B; if symptoms have disappeared, continue plan A.					
6th to 9th	Sulfathiazole	2.0	30	0.5	7½	Every 5 h., 6 AM to 9 PM
10th	If patient remains symptom-free, stop all treatment.					
10th to 14th	None					
14th	Proceed with determination of cure.					

2. Plan B.—To be used in sulfathiazole-resistant patients on 5th day.

Day or days (incl.)	Drug	Daily dosage		Single dose		Time of administration
		Gm	*Gr*	*Gm*	*Gr*	
5th	Sulfapyridine	3.0	45	0.5	7½	Every 3 h., 6 AM to 9 PM
6th to 9th	Sulfapyridine	2.0	30	0.5	7½	Every 5 h., 6 AM to 9 PM
10th	Examine patient; if discharge persists, stop sulfapyridine and evacuate patient to hospital for special care; if symptoms have disappeared, continue plan B.					
10th to 15th	Sulfapyridine	2.0	30	0.5	7½	Every 5 h., 6 AM to 9 PM
16th	If patient remains symptom-free, stop all treatment.					
16th to 20th	None					
20th	Proceed with determination of cure.					

d. Determination of cure (male).—On the 14th to 20th day after the start of treatment, and if all symptoms have disappeared—

(1) Before patient voids, massage the prostate with sufficient vigor to obtain secretion.

(2) Examine a gram-stained smear of the secretion for pus and gram-negative intracellular diplococci.

(3) If no diplococci are found, repeat this procedure weekly for 8 weeks; if negative for 8 weeks, discharge the patient as cured; supporting evidence of culture is desirable (see below).

(4) If, on any of these occasions, the prostatic secretion contains diplococci by smear or gonococci by culture, repeat plan A (*c*(3)(*f*)*1* above), using sulfapyridine instead of sulfathiazole

(dosage identical); and in addition *gently* massage the prost twice weekly.

(5) Provocative tests, such as the passage of instruments i the urethra, indulgence in alcohol, and sexual excitement, *not* recommended as criteria of cure.

e. Determination of cure (female).—(1) Do a pelvic exami tion to determine the presence of masses or discharge.

(2) Examine gram-stained smears from the urethra o every 2 weeks for 3 months.

(3) Confirm negative or positive urethral smears by cult from the urethra and cervix at monthly intervals on last d of the menstrual period. To obtain material for smear a culture—

(*a*) Massage the urethra and Skene's glands.

(*b*) Use a bivalve speculum to expose the cervix (do not a lubricant on the speculum).

(*c*) Cleanse the external os with dry cotton.

(*d*) Squeeze the cervix gently with blades of the specul removing any material obtained with the blades of the specul

(*e*) Remove the material from the speculum with a platin loop or cotton-wound applicator for smear; use a cotton-wou applicator for culture.

(4) If no diplococci are found by smear or gonococci by c ture during 3 or 4 months of post-treatment observation, d charge the patient as cured.

(5) If, on any of these occasions, diplococci are found smear or gonococci by culture, repeat plan A, using sulfapyrid instead of sulfathiazole.

f. Cultures in determination of cure.—Absolute proof of c in gonorrhea demands a minimum of three consecutive ne tive cultures of prostatic secretion in men (at 2-week interval or of secretions from the cervix and urethra in women (at oftener than monthly intervals). If cultures are not availab determination of cure must depend on gram-stained smears.

g. Local treatment in sulfonamide-resistant cases or th with complications arising during treatment (posterior ureth tis, prostatitis, etc.).—When local treatment is necessary, should be given at dispensaries or hospitals by specially trai personnel.

(1) For sulfonamide-resistant cases of anterior urethriti

(*a*) Daily, until discharge ceases, *gently* irrigate the anter

urethra with 1,000 cc of a warm 0.02 percent solution of potassium permanganate by the gravity method, with the reservoir not more than 3 feet above the meatus; do not distend the urethra.

(*b*) Daily, until discharge ceases, inject not more than 8 cc of a 5 or 10 percent solution of mild silver proteinate (not more than 1 week old) into the anterior urethra; retain for 3 minutes by having the patient compress the meatus with the thumb and finger.

(*c*) All irrigations and instillations should be administered by a medical officer or trained attendant, *not by the patient.*

(2) For the active stage of posterior urethritis occurring as complication of treatment—

(*a*) Stop all local treatment until the acute symptoms have subsided.

(*b*) Give hot hip baths for painful urination.

(*c*) Resume daily irrigations of the *anterior* urethra with a 1:2 percent solution of potassium permanganate when acute symptoms have subsided.

(*d*) After a week during which the first glass of urine has been nearly clear and the second glass entirely so, gently stroke the prostate before the patient voids; if this causes a persistent urethral discharge or results in symptoms of active posterior infection, do not repeat until the patient has been free of symptoms and the first glass of urine has been nearly clear and the second glass entirely so for 1 week.

(*e*) If the first massage does not cause any activation of the signs or symptoms, repeat massage of the prostate twice a week, and increase the pressure gradually until it is fairly firm; continue the prostatic massage until the prostatic secretions become negative for gonococci or diplococci.

(*f*) Make microscopic studies of the prostatic secretion once a week for at least 8 weeks; if all are negative, discharge the patient as cured. (Cultures of prostatic secretions should be made if facilities are available.)

(3) All cases of posterior urethritis, epididymitis, acute prostatitis, acute seminal vesiculitis, and acute arthritis arising as complications of treatment require hospital care; if one of these complications is the original manifestation that brings the patient under medical observation, it can frequently be controlled by a day or two of ambulatory chemotherapy.

(4) Chronic prostatitis, symptomless and manifested only by pus in the prostatic secretion should, in general, be allowed to go untreated; prolonged courses of prostatic massage are valueless.

h. Serologic follow-up of patients with gonorrhea.—So far as possible, all patients, male and female, acquiring acute gonorrhea should have a serologic test for syphilis done on admission and at least one follow-up test to rule out symptomless infection with the latter disease; if only one such test is done, it should be performed 3 or 4 months after the onset of gonorrhea.

38. Syphilis.—*a. General procedures of treatment.*—(1) No treatment is to be given for suspected early syphilis until the diagnosis is made either by darkfield examination of by confirmed serologic tests; no therapeutic tests are to be used.

(2) Begin with an arsenical in early and latent syphilis (one-half full dose initially, followed by full doses); begin with bismuth in late syphilis.

(3) The preferred arsenical outside a hospital is arsenoxide (mapharsen); when mapharsen is not available, neoarsphenamine may be used; the preferred bismuth preparation is bismuth subsalicylate; in hospitals under competent direction arsphenamine or other arsenicals may be employed.

(4) Tryparsamide and fever therapy are not to be used outside a hospital.

(5) The range of dosage of arsenoxide (mapharsen) is 0.01 to 0.06 gram per intravenous injection, of neoarsphenamine, 0.45 to 0.90 gram, of arsphenamine, 0.3 to 0.6 gram; the average dose of bismuth subsalicylate is 0.2 gram.

(6) A course of an arsenical is eight weekly intravenous injections; a course of bismuth is ten weekly intramuscular injections; a normal rest period, when allowed, is 8 weeks.

(7) Treatment is to be of the continuous alternating type, that is, without rest periods between courses and with arsenical courses alternating with bismuth courses—not alternate injections or simultaneous injections of the two drugs except as indicated for overlap.

(8) Each treatment is to be recorded on the syphilitic register of the patient, or if for any reason a syphilitic register is not available, a written record is to be kept and transferred to the standard form as soon as possible.

(9) Each entry should include date, serial number of injection, drug, dose, and reaction.

(10) It cannot be too strongly emphasized that clock-like regularity of the treatment schedule, without long or short time variations or lapses, is critically important to both infection control and cure, being most important in the first 12 months; every effort must be made to impress this fact on enlisted men and officers, as well as on the medical personnel, on all occasions.

(11) Emphasis should also be placed on the completion by each patient of the *full* schedule of treatment in the time called for by the type of infection presented, discarding serologic tests as guides, and so-called "abortive" procedures in seronegative primary syphilis or "treatment to noninfectiousness."

(12) The infectiousness of syphilis is *not* to be predicated on a blood test result, but on the total time, course, laboratory tests, physical inspection, and treatment summation of the case.

b. Serologic controls in treatment.—(1) Early syphilis (first 2 years of infection) is to have a blood serologic test—

(*a*) At the start of treatment.

(*b*) At the start of the second course of treatment, if negative at the beginning.

(*c*) At the 16th week (the average positive case should become seronegative).

(*d*) At the 24th week (a positive reaction indicates need for spinal fluid examination).

(*e*) At the beginning and end of each bismuth course thereafter.

(*f*) Once in 3 months during the 2 years of post-treatment probation.

(*g*) Before discharge from the service at any time.

(2) Early syphilis is to have a spinal fluid test—

(*a*) Between the 24th and 52d weeks in all cases.

(*b*) If the fluid is abnormal, at least once every 6 months.

(*c*) If weak or strongly positive blood tests appear after negative ones.

(3) Early latency (first 4 years of the infection) is to be considered early syphilis in serologic controls both as to blood and as to spinal fluid.

(4) Late latency (after 4th year or infection of uncertain duration) is to have a blood test once in 3 months whether on

treatment or probationary observation (30 percent irreversible are to be expected).

(5) Latent syphilis is to have a spinal fluid test—

(*a*) As soon after treatment is begun as practicable.

(*b*) If, after a series of negative tests, the blood becomes positive.

(*c*) Before discharge, if practicable, unless previously negative.

c. Control or relapse and infectiousness.—(1) Early syphilis is to be regarded as infectious until the second series of injections has been given, and should be rated potentially infectious if treatment for any reason is irregular or inadequate (sub schedule).

(2) Physical inspection of the skin (including the palms and soles), mucous membranes, anus, and genitalia should be performed as often as circumstances permit during treatment and at each probationary inspection.

(3) Patients should be warned to look for and report mouth, skin, and genital lesions.

(4) The involution of the chancre or secondary lesions should be watched, to detect treatment-resistant cases.

(5) Darkfield examination is of great help in recognizing skin, mucosal and genital relapse lesions; the blood test may be negative.

(6) Relapsing or treatment-resistant infectious types of cases require special consideration with a view to effective therapy and quarantine control.

d. Treatment interpretation of spinal fluid findings.—(1) *Spinal fluid reports.*—These should include—

(*a*) Quantitative complement-fixation test.

(*b*) Total protein or standard globulin test.

(*c*) Differential cell count.

(*d*) Colloidal gold (gold-sol) test (the fluid should be bloodless).

(2) *Minimal abnormality.*—Slight increase in protein (pandy +— to +) or leukocyte count over 5. Continue with standard treatment, recheck at or before probation.

(3) *Grade I.*—Blood test positive or negative; spinal fluid Wassermann test negative; leukocyte count 10 to 25; protein + colloidal gold test low second zone (syphilitic). The patient will recover under continuance of standard treatment; recheck in 6 months.

(4) *Grade II.*—Blood test positive or negative, usually positive; spinal fluid Wassermann test positive in low dilutions, negative in high dilutions; leukocyte count 5 to 100 or more; protein ++; colloidal gold test pronounced second zone (syphilitic). The patient will recover under prolongation and intensification of standard intravenous and intramuscular treatment.

(5) *Grade III.*—Blood test strongly positive; spinal fluid Wassermann test strongly positive in all dilutions; leukocyte count 7 to 100 or more; protein +++; colloidal gold test first zone (paretic). The patient will not recover without tryparsamide or fever therapy or both; he should be sent to a hospital as soon as practicable.

e. Reaction prevention.—(1) *Examination.*—A physical examination and a urine examination should precede the institution of treatment.

(2) *Inspection.*—A minimal medical inspection should be made before each treatment; the *neck and arms should be exposed.* Inspect—

(*a*) Face, for edema of eyelids and dermatitis.

(*b*) Mouth, for bleeding gums and petechiae.

(*c*) Scleras, for jaundice.

(*d*) Elbow flexures and wrists, for dermatitis and petechiae.

(*e*) For signs of intercurrent infection or fever. (Any positive finding demands closer examination.)

(3) *Questions.*—Sick after last treatment? Bowels loose? Urine dark? Skin itch? Any rash? Gums or nose bleed? Stool black? Pain in arm or hip? (These questions may, if practicable, be posted in sight of the patient and pointed to by the person giving the treatment.)

(4) *Technical suggestions.*—(*a*) Discard discolored drugs and solutions and damaged ampules.

(*b*) Shake and aerate arsenoxide; do not shake or aerate the other arsenicals.

(*c*) Inject arsenoxide rapidly to avoid thrombosis (no danger of speed shock or nitritoid crisis); other arsenicals should be injected *slowly* to avoid speed shock or nitritoid crisis.

(*d*) Thoroughly shake oily suspensions of bismuth.

(*e*) Aspirate with syringe after insertion of the needle before injecting anything, especially intramuscularly.

(*f*) Inject bismuth intramuscularly into the inner angle, upper outer quadrant of buttock; alternate.

(*g*) Massage long and firmly after withdrawing the needle from the buttock.

(*h*) Have the patient prolong massage to 3 minutes.

(*i*) Rest, if practicable, after arsenicals; exercise after intramuscular injections.

(*j*) Warn the patient to report his reactions.

(*k*) Test the urine once in 2 to 4 weeks; do not stop or modify treatment for slight albuminuria or cylindruria.

(*l*) Watch the mouth and gums; prescribe a mouth wash of hydrogen peroxide or other oxidant and an astringent wash; give indicated dental attention.

f. Reaction therapy.—(1) For speed shock or nitritoid crisis inject subcutaneously 0.5 to 1.0 cc of a 1:1000 solution of epinephrine (adrenalin).

(2) For jaundice or dermatitis, inject intravenously 500 cc of a 5 percent solution of glucose.

(3) For threatened cerebral accidents, venesect (400 cc).

(4) Liver extract intramuscularly is occasionally helpful in suspected liver damage.

(5) For cerebral vascular accidents, inject intravenously 500 cc of a hypertonic (1.5 percent) saline solution.

(6) For blood dyscrasias give transfusions.

(7) The value of sodium thiosulphate for any type of treatment reaction is questionable.

(8) Treatment should be stopped and the patient hospitalized if the following appear:

(*a*) An itchy dermatitis of the face and flexures.

(*b*) Jaundice.

(*c*) Petechiae or other hemorrhagic lesions.

(*d*) Evidence of cerebral injury, even though slight.

(9) If treatment is stopped because of reaction, medical consultation as to further procedure should be had as promptly as possible, especially in patients with early syphilis.

g. Cardiovascular, visceral, and resistant neurosyphilis.—Cardiovascular, visceral, and symptomatic and asymptomatic neurosyphilis with a Grade III spinal fluid require special treatment in a hospital. (For additional details see standard texts.)

h. Precocious late syphilis (tertiarism).—Patients with precocious late syphilis (early gummatous and rupial lesions and bone lesions following lapse after inadequate heavy-metal cover-

age of arsenical treatment) should be hospitalized for combined fever and arsenical therapy as soon as possible.

i. Congenital syphilis.—On the recognition or appearance of active lesions, congenital syphilis should be treated on the schedule of early syphilis.

j. Diagnostic nomenclature for syphilis and its complications in the Army.—(1) The following diagnostic terms should be used on all records:

Primary.
Secondary, early.
Secondary, relapsing.
Latent.
Tertiary—
    Mucocutaneous.
    Osseous.
    Ocular.
    Visceral.
    Other (specify).
Cardiovascular—
    Aneurysm (saccular).
    Aortic regurgitation.
    Aortitis, uncomplicated.
Neurosyphilis—
    Asymptomatic.
    Acute syphilitic meningitis.
    Diffuse meningovascular.
    Tabes dorsalis.
    Taboparesis.
    General paresis.
    Gumma.
    With psychosis (other than paresis).
Congenital.
Type undetermined.
Poisoning from arsphenamine (includes all arsphenamines and mapharsen); specify nature, as—
    Jaundice.
    Dermatitis.
    Blood dyscrasia.
    Hemorrhagic encephalitis.
    Other (specify).

Poisoning from tryparsamide; specify nature, as—
Amblyopia.
Dermatitis.
Other (specify).
Poisoning from bismuth (specify manifestation).
Poisoning from mercury (specify manifestation).
Poisoning from iodides (specify manifestation).
Fever therapy for syphilis—
Malaria inoculates.
Mechanical fever.
Other (specify).
No disease, spinal puncture.

(2) The terms are used as follows:

(*a*) *Primary.*—To include those cases presenting the prima lesion of syphilis (chancre) which have not yet develop secondary manifestations. This diagnosis must be confirmed darkfield examination, serologic test of the blood, or *both.* the blood seroligic test is negative, the diagnosis of prima syphilis is not permissible without the demonstration of *Trep nema pallidum* by darkfield.

(*b*) *Secondary, early.*—To include only those cases of ear syphilis which show one or more of the manifestations of sy temic dissemination of the spirochete; for example, generalize enlargement of the lymph nodes, cutaneous eruption, mucou patches, condylomas, patchy alopecia, laryngitis, bone pains, febrile reaction. The chancre may or may not be present an if present, may be in any stage of evolution. In early secondar cases the manifestations of systemic spirochetal disseminatio are increasing, have attained their maximum, or are waning This diagnosis must be confirmed by darkfield examination serologic test, or both. Ocular or neurologic complication (iritis, neuroretinitis, and acute syphilitic meningitis ma occur, and should be specially recorded as: "Syphilis, secondary early. Manifested by ____."

(*c*) *Secondary, relapsing.*—In relapsing secondary syphilis, second systemic dissemination of the spirochete has taken place usually because of premature cessation of arsenical therapy The commonest time for relapse is within the first 6 months and the majority of all early relapsing cases occur within th second year. In addition to relapsing lesions of the skin an mucous membranes that may be observed, others may also occur

particularly in the eyes and in the nervous system. These cases should be reported as: "Syphilis, secondary, relapsing. Manifested by _____."

(*d*) *Latent.*—Secondary symptoms have subsided, and the active manifestations of late syphilis have not yet supervened. There are no evidences of syphilis other than a positive blood serologic test. Cases should not be classified as latent unless asymptomatic neurosyphilis has been excluded by a negative examination of the spinal fluid. The date of the negative examination of the spinal fluid should be stated in all such cases, as follows: "Spinal fluid negative (date)." Cases which show no evidence of syphilis other than a positive blood serologic test and in which involvment of the central nervous system has not yet been excluded by examination of the spinal fluid are to be reported as: "Syphilis, type undetermined, spinal fluid not examined. Manifested by _____."

(*e*) *Tertiary.*—(1) This classification is to be limited to cases that show active lesions of late syphilis other than involvment of the central nervous system and the cardiovascular system. The lesion may be a gumma or it may be a diffuse process, and may involve any organ or tissue of the body. If the central nervous system or the cardiovascular system is involved, however, these cases should not be reported as tertiary syphilis but, because of their gravity and frequency and the necessity for specialized treatment should be reported as neurosyphilis or as cardiovascular syphilis, as the case may be.

(2) The majority of patients with tertiary syphilis, other than cardiovascular syphilis or neurosyphilis, fall within four categories: mucocutaneous (late syphilitic gummatous lesions of the skin or mucous membranes); osseous (periostitis, osteomyelitis, arthritis, and synovitis); ocular (iritis, uveitis, keratoiritis, keratitis, and choroiditis, but not including optic atrophy); visceral (hepatic and gastric). Such cases should be specially diagnosed as follows: "Syphilis, tertiary, mucocutaneous;" "syphilis, tertiary, osseous;" "syphilis, tertiary, ocular;" "syphilis, tertiary, visceral." If tertiary manifestations occur which do not fit into one of these categories, diagnose as: "Syphilis, tertiary, other (specify)."

(*f*) *Cardiovascular.*—To include all lesions of the heart and great vessels.

(*g*) *Aneurysm* (*saccular*).—Do not use for a fusiform dilatation of the aorta. Specify artery involved.

(*h*) *Aortic regurgitation.*—Specify whether with or without cardiac decompensation.

(*i*) *Aortitis, uncomplicated.*—To be used only for those patients with symptoms and physical or X-ray signs of syphilitic aortic involvement in the absence of aneurysmal sacculation or aortic regurgitation.

(*j*) *Neurosyphilis.*—To include all cases with involvement of the central nervous system.

(*k*) *Asymptomatic.*—To be used only for those patients with early or late syphilis who have no symptoms or detectable physical signs of central nervous system involvement, and in whom the diagnosis is based on the routine finding of abnormalities in the spinal fluid.

(*l*) *Acute syphilitic meningitis.*—Usually occurs within the first 2 years of the disease, most commonly as a relapse phenomenon (neurorecurrence), manifested by the usual signs of low grade meningeal involvement, with or without cranial nerve palsies.

(*m*) *Diffuse meningovascular.*—This is a catch basket category to include all cases of neurosyphilis that do not fit into other diagnostic categories enumerated; the manifestations should be stated.

(*n*) *Tabes dorsalis.*—The manifestations should be stated.

(*o*) *Taboparesis.*—To be used only in patients with definite psychiatric signs of paresis complicated by definite clinically demonstrable evidence of damage to the posterior columns of the spinal cord.

(*p*) *General paresis.*—To be limited to cases which show psychic changes in addition to neurologic signs and the characteristic changes in the spinal fluid. Patients with a paretic-type spinal fluid but without psychic changes should be reported as: "Syphilis, diffuse meningovascular. Manifested by ------." or as some other type of neurosyphilis.

(*q*) *Gumma.*—This should only include gummas of the brain and spinal cord. Gummas of other organs or tissues to be reported as: "Syphilis, tertiary. Manifested by ------."

(*r*) *With psychosis.*—To include neurosyphilis with psychosis other than cases of paresis and taboparesis.

(*s*) *Congenital.*—To be limited to cases that show definite evidence of the existence or former existence of the characteristic changes of congenital syphilis, such as interstitial keratitis, Hutchinson's teeth, saber shins and other bone changes, saddle nose, 8th nerve deafness, etc. (The congenital origin of syphilis is not to be assumed merely because the time and circumstances of the infection cannot be ascertained and because there is no scar of a primary lesion.)

(*t*) *Type undetermined.*—To include cases in which accurate diagnosis has not been made. (Every effort should be made to make a complete examination and proper diagnostic classification in all cases.)

(*u*) *Poisonings.*—The suggested term for various drug poisonings are intelligible as they stand.

(*v*) *Fever therapy.*—It is particularly desirable to diagnose those patients treated with fever, and to specify the type of fever employed.

(*w*) *No disease, spinal puncture.*—This should be used in all cases, syphilitic or otherwise, routinely hospitalized for the purpose of spinal puncture except where pertinent diagnoses are in order. In syphilitic cases record the result of the spinal fluid test as positive or negative.

(3) The manifestations of the disease should be given in each case. Lesions should be located and briefly described. Results of laboratory procedures should be stated.

(4) The diagnosis of syphilis should not appear in the record of any person unless the presence of the disease has been established. For that reason cases not definitely so diagnosed should *not* be recorded *at any time* as: "Under observation for syphilis."

(5) When syphilitic patients under treatment or during post-treatment observation are admitted to or retained in a hospital for follow-up examination (including spinal puncture), and when no new developments are found that justify a change or addition to the original diagnosis as to syphilis, the diagnosis for that hospital admission or portion thereof due to observation for syphilis shall be recorded as syphilis, old, of the type in existence at the time of admission or retention in the hospital, unless all tests for syphilis are negative and the patient can be pronounced cured, in which latter case the diagnosis shall be recorded as: "Under observation (syphilis); no disease."

39. Chancroidal infection.—*a. Definition.*—Chancroid is a venereal disease transmitted only by direct contact and characterized by single or multiple genital ulcers possessing irregular crateriform margins, usually nonindurated bases, and a tendency toward the formation of a complicating suppurating inguinal adenitis; the incubation period is usually 3 to 14 days.

b. Diagnosis.—It is important to rule out the presence of mixed syphilitic and chancroidal infection. For this purpose all local medication should be withheld until three negative dark-field examinations, carried out on successive days, have been obtained from accessible lesions. During this period, saline dressings alone should be used. A blood serologic test for syphilis must be secured, and the Frei test for lymphogranuloma venereum should be carried out, if possible. *Blood tests for syphilis should be made at monthly intervals for 6 months following healing of the chancroidal lesions.* Laboratory tests for the absolute diagnosis of chancroid (Ito-Reenstierna skin test or the staining or cultural isolation of the Ducrey bacillus) are *not* recommended.

c. Treatment.—(1) *Chemotherapy*—(*a*) *Local.*

1. Accessible lesions should be cleansed with soap and water, and dried.

2. They should then be completely covered with powdered sulfanilamide, and a loose, dry dressing applied; this should be repeated at daily intervals until the lesion heals.

3. In patients with tight phimosis and underlying ulcerative lesions, the phimotic preputial cavity should be irrigated twice daily with a 0.02 percent solution of potassium permanganate.

(*b*) *Systemic.*

1. Administer 3.0 grams (45 grains) of sulfanilamide daily for 5 days, utilizing divided doses at 4-hour intervals.

2. For the 9 succeeding days administer 2.0 grams (30 grains) sulfanilamide daily, also in divided doses.

3. At the end of 14 days, discontinue all chemotherapy.

Practically all chancroidal infections will respond to the above routine. In fact, if the lesion does not heal, doubt is cast on the correctness of the diagnosis, and the patient should be

restudied from the diagnostic standpoint, and, if necessary, treated surgically.

(2) *Surgical therapy.*—(*a*) Surgical procedure designed to relieve phimosis or paraphimosis should be resorted to only on the basis of sound clinical judgment.

(*b*) Most chancroidal buboes subside with systemic sulfanilamide therapy; if extensive suppuration is already present or occurs, the bubo may be opened by a small incision, the pus aspirated, and the cavity packed with sulfanilamide powder.

40. Lymphogranuloma venereum.—*a. Definition.*—This disease concept includes the conditions formerly known as lymphogranuloma inguinale, lymphopathia venereum, climatic bubo, esthiomene, and inflammatory rectal stricture.

b. Etiology.—It is caused by a filterable virus, which probably has multiple strains.

c. Geographic distribution.—The disease is world-wide, but most frequent in the Tropics.

d. Clinical picture.—It is a systemic disease of the lymphatic vessels and structures, usually originating in a trivial and transitory lesion of the penis, vulva, vagina, or rectum, which frequently escapes the patient's notice. The invasion of the lymph nodes usually occurs from 10 to 30 days after infection, and occasionally is delayed for months. The inguinal adenitis is often bilateral, and occasionally subsides without suppuration. During this stage, constitutional symptoms may be observed. Lymph nodes may fuse to the skin, resulting in multiple areas of softening, followed by numerous fistulas. Extensive scarring accompanies healing. The anorectal syndrome is usually found in women, and is characterized by rectal pain, discharge of blood and pus from the anus, a tendency toward extreme chronicity, and the production of rectal stricture.

e. Diagnosis.—The disease should be differentiated from malignant tumors, Hodgkin's disease, tularemia, tuberculosis, pyogenic infections, chancroidal bubo, and syphilis.

f. Specific diagnosis (Frei test).—(1) In an infected case, intracutaneous injection of 0.1 cc Frei antigen gives rise to an inflamatory papule at least ¼ inch in diameter, often with peripheral erythema and sometimes a central vesicle; the papule appears within 48 hours, and persists for several days.

(2) In infected persons, the Frei test may give positive results for years.

(3) In suspected cases, negative tests should be repeated; after the development of the bubo, positive reactions are obtained in over 95 percent of cases.

(4) Mixed venereal infections should be ruled out by the dark-field examination of material from genital lesions for the causative organism of syphilis; frequent serologic examinations should be continued for at least 6 months after the disappearance of the lymphatic symptoms.

g. Treatment.—(1) *Local.*—(*a*) Patients with acute inguinal adenitis should be hospitalized whenever possible; the fluctuant nodes may be aspirated, but incision and drainage should be delayed until the effect of chemotherapy has been observed.

(*b*) Patients with the acute anorectal syndrome should be treated in the same manner.

(*c*) Patients with rectal stricture are problems for hospital management.

(2) *Chemotherapy.*—(*a*) The value of the sulfonamide compounds in the treatment of lymphogranuloma venereum has not yet been established, but there are some clinical indications that they may be useful. If employed, the following schedules may be followed:

1. Sulfanilamide.—Sulfanilamide therapy should consist of the administration of 1.25 grams (20 grains) of the drug four times daily for the first 4 or 5 days, followed by a reduction to 0.50 to 0.75 gram (7½ to 12 grains) four times daily for an additional 7 days; if inflammatory symptoms (not residual scarring) persist, repeat the chemotherapeutic course after a rest period of 10 to 14 days, utilizing the same or one of the allied chemotherapeutic agents.

2. Sulfapyridine.—Sulfapyridine should be administered in dosage of 3 grams (45 grains) for the first day and 2 grams (30 grains) for the following 10 to 12 days (in divided doses at 4-hour intervals); the course should be repeated following a rest period of 10 to 14 days if clinical evidence indicates active extension of the lymphatic process.

(*b*) The acute anorectal syndrome may be treated in the same manner; stricture or other late complications should receive special consideration.

(3) *Biologic treatment.*—(*a*) Frei antigen given intravenously in 0.2 to 0.3 cc amounts every second day may be of value.

(*b*) The duration of treatment depends on the patient's response.

41. Granuloma inguinale.—*a. Definition.*—Granuloma inguinale is a chronic disease due to infection with a leishmania-like organism. It primarily involves the skin and mucous membranes, rarely with coincident adenopathy, and is characterized by vivid-hued, shining, verrucous, vegetating nodules of granulating tissue with a hemorrhagic surface surrounded by a thin, easily excoriated epidermis. The condition spreads by peripheral extension and autoinfection, often involving the entire genital area. It may involve large adjacent areas of the lower abdomen and thighs. The lesions show little or no tendency to spontaneous healing and may persist for months or years.

b. Diagnosis.—(1) The clinical appearance of a chronic process involving the groin and genital areas with little involvement of the lymph nodes is characteristic of the disease.

(2) The finding of Donovan bodies in Wright-stained smears of deep-tissue scrapings or in biopsy specimens of a peripheral area of disease tissue (including a section of normal adjacent skin) confirms the diagnosis.

(3) Lymphogranuloma venereum, chancroid, and syphilis should be considered in the differential diagnosis, and the appropriate test for each condition should be performed.

c. Treatment.—(1) Tartar emetic should be administered intravenously in doses of 0.03 to 0.12 gram (beginning with 3 cc and increasing to 12 cc, if tolerated, of a 1 percent solution three times a week), the maximum tolerated dose to be given for fifteen doses.

(2) One to 3 cc (0.06 to 0.18 gram) of Fuadin or 1 to 3 cc (0.06 to 0.18 gram) of Anthiomaline (both of these are complex antimony compounds) may be given intramuscularly two or three times weekly for twenty or twenty-five doses when the patient has difficulty in taking tartar emetic, or when the lesions have not improved satisfactorily under the former drug.

(3) Tartar emetic, Fuadin, or Anthiomaline should be continued at weekly intervals for at least 4 month after all lesions are completely healed, otherwise relapse is almost certain to occur.

(4) Local treatment of the lesions is often limited to daily dressings; however, surgical excision of the entire area may be necessary. Large areas may be treated with pencils of carbon dioxide snow.

SECTION V

CHEMOTHERAPY AND SEROTHERAPY IN CERTAIN INFECTIOUS DISEASES

42. General.—*a.* This section, covering the chemotherapy and serotherapy of certain infectious diseases, is based on the recommendations of the Committee on Chemotherapeutic and Other Agents, Division of Medical Sciences, National Research Council, as published in Circular Letter Number 81, Office of The Surgeon General, December 5, 1940, and recent revisions. Because of the rapid development of chemotherapeutic agents, some of these recommendations have already been changed, and others are likely to be modified from time to time. Certain other measures are described briefly where it seems appropriate. However, it is impracticable, except in a broad way, to delineate the therapy of individuals suffering from various infections. The following paragraphs, therefore, serve as a general guide for medical officers and are to be used at their discretion, with due consideration of all other factors that may be presented by each individual case. Other indicated therapeutic or nursing procedures should not be excluded or neglected.

b. The Medical Department will continue to supply therapeutic sera for patients who are sensitive to chemotherapeutic agents (for toxic effects see *c*(6) below), and for those who, in the opinion of the medical officer, may be advantageously treated with such sera.

c. The following general information applies to the administration and precautions in the employment of compounds of the sulfonamide group, and instructions for the preparation of solutions for parenteral administration:

(1) *Sensitivity.*—In view of occasional *sensitivity* to the sulfonamide compounds, these drugs should not be administered until after careful interrogation concerning previous medication with sulfonamides and any ill effects which may have been experienced by the patient. In case sensitivity to these drugs is suspected, a small test dose (0.1 to 0.3 grams) should be given and the patient observed for 12 hours before intensive therapy is started. Following this the patient should be carefully observed and the drug immediately stopped on the first appearance of any toxic manifestation.

(2) *Previous medication.*—In occasional instances it will be found that upon admission a patient has been taking one or more of the sulfonamide drugs, either by self-administration or during the ambulatory phase of his disease upon orders of his attending physician. All patients should be carefully questioned regarding previous medication because large initial dosage may result in an excessive amount being given. Where clandestine self-administration is suspected, or where previous medication in an unknown quantity has been given, a preliminary determination of the blood concentration should be made, and the initial dosage determined accordingly.

(3) *Indiscriminate medication with sulfonamides.*—There is a tendency to administer drugs of the sulfonamide group as a cure-all in minor illnesses, and in illnesses where the indications for their use are not clear. It must be realized that these drugs are not without certain hazards of administration and their use should be carefully controlled.

(4) *Supplementary drugs.*—The routine use of bicarbonate of soda is unnecessary in *sulfonamide* therapy.

(5) *Preparation of parenteral solutions.*—(*a*) Intravenous solutions, except in the case of sulfanilamide, are made up *only* with the *sodium salts* of sulfonamides. There is *no sodium salt of sulfanilamide* and the drug is *rarely* given intravenously. The sodium salts are prepared in *5 percent solutions.* To make a 5 percent solution of sodium salts measure 5 grams (75 grains) of the salt, transfer it from the bottle with a sterile spatula and weigh it out in a sterile container, then add to 100 cc of sterile

freshly distilled water. These solutions cannot be boiled or autoclaved. They are alkaline, the alkalinity being sufficient to kill all bacteria except spores. Because of alkalinity, these solutions must be given intravenously *slowly* (15 minutes), care being taken not to get the material outside the vein because if this occurs it may cause a slough. For this reason, administration by the subcutaneous or the intrathecal route should be avoided. These solutions should *never* be added to transfusion blood, saline, glucose, or intravenous fluids.

(*b*) To prepare a 1 percent solution of sulfanilamide, 1 gram of crystalline sulfanilamide should be measured, using sterile precautions as in (*a*) above, and transferred to 100 cc of freshly prepared sterile *normal saline* solution which has been brought to a boil. This solution is practically always given by the subcutaneous route, rarely by the intravenous route.

(6) *General comment on toxic effects.*—(*a*) It is to be recommended, when possible, that laboratory control of sulfonamide chemotherapy be carried out, but inability to do blood and urine examinations should never be considered a contraindication to the use of these drugs. However, when facilities are available, the laboratory procedures outlined in (7) below should be carried out in patients who are receiving sulfonamide drugs. Patients receiving sulfonamide compounds should be seen at least once a day and should be questioned as to the presence of headache or malaise. These are frequently important early symptoms of toxic reaction. Patients should be inspected at each visit for the presence of jaundiced sclerae (acute hemolytic anemia or hepatitis), pale mucous membranes (acute hemolytic anemia), or rash. The temperature should be carefully recorded. *With recurrent fever after normal temperature in the course of sulfonamide therapy, the drug should be discontinued immediately or if recently discontinued, should not be resumed unless it has been demonstrated that the fever is due to a recurrence of the infection.* Whenever therapy with the sulfonamide drugs is stopped because of a drug reaction, *fluids should be forced to 5,000 cc per day in order to wash out the drug.*

(*b*) Agranulocytosis is extremely rare before the 14th day of therapy. *It is imperative that total and differential white blood cell counts be made in patients still under treatment after the 10th day,* every 2 days from the 14th to the 40th day. If the polymorphonuclear leukocytes fall to 50 percent or less in

adult patients, stop the drug immediately. Granulocytopenia without agranulocytosis may occur.

(*c*) Any patient who has had a toxic reaction to one of the sulfonamide group of drugs may have a second and more severe toxic reaction if one of these drugs is prescribed again. In such individuals a small test dose of the drug (0.1 to 0.3 grams) should be given and the patient observed for 12 hours before intensive therapy is started following which the patient must be carefully observed and the drug immediately stopped on the first appearance of any toxic manifestation.

MANIFESTATIONS OF DRUG TOXICITY NOTED IN ADULTS TREATEDWITH SULFANILAMIDE, SULFAPYRIDINE, SULFATHIAZOLE, SULFAGUANIDINE† OR SULFADIAZINE

Reaction	Sulfanilamide	Sulfapyridine	Sulfathiazole	Sulfadiazine
Nausea, vomiting	Fairly common	Frequent	Uncommon	Uncommon.
Dizziness	Common	Common	Uncommon	Uncommon.
Psychoses*	0.6 percent; occur early	0.3 percent; occur early	Rare	Questionable.
Neuritis**	Very rare	Not reported	Rare	Not reported.
Cyanosis	Very common, early, and late	Faint, common, early, and late	Uncommon	Rare.
Acidosis*	1.9 percent; occurs at any time	None	None	None.
Fever*	10 percent; generally 5th to 9th day; may occur 1st to 30th day.	4 percent; generally 5th to 9th day; may occur 1st to 30th day.	10 percent; generally 5th to 9th day.	Uncommon; 1 percent.
Rash*	1.9 percent; may take any form, generally 5th to 9th day; may occur 1st to 30th day.	2 percent; may take any form, 5th to 9th day; may occur 1st to 30th day.	5 percent; nodular type, common; may take any form, 5th to 9th day.	Uncommon; 2 percent.
Hepatitis**	0.6 percent; early or late	Not seen, but reported	Rare	Not reported.
Leukopenia with granulocytopenia.**	0.3 percent; early or late	0.6 percent; early or late	1.6 percent; early or late	Rare.
Acute agranulocytosis**	0.1 percent; occurs 14th to 40th day; common 17th to 25th day.	0.3 percent; occurs 14th to 40th day; common 17th to 25th day.	Reported	Reported.
Mild hemolytic anemia	3 percent; early or late	Rare	Rare	Rare.
Acute hemolytic anemia**	1.8 percent; occurs 1st to 5th day.	0.6 percent; occurs 1st to 5th day.	Very rare	Very rare.
Hematuria*	Not reported	8 percent; generally early	2.5 percent; generally early	1 percent.
Anuria with azotemia**	Not reported	0.3 percent; generally 1st 10 days.	0.7 percent; generally 1st 10 days.	Rare.

Hyperleukocytosis*	Generally in presence of acute hemolytic anemia.	Generally in presence of acute hemolytic anemia.	Not reported	Not reported.
Injection of sclerae and conjunctivae.**	Not reported	Not reported	4 percent; may occur with rash and fever, 5th to 9th day.	Rare; 0.3 percent.
Purpura hemorrhagica**	Not seen, but reported	Not seen, but reported	Not reported	Not reported.
Ocular and auditory disturbances.**	Rare	Rare	Very rare	Not reported.
Jaundice**	With acute hemolytic anemia or hepatitis.	With acute hemolytic anemia or hepatitis.	With acute hemolytic anemia or hepatitis.	Not reported.
Painful joints*	Reported	Not reported	Reported with rash, etc	Not reported.
Stomatitis*	Rare	Not reported	Not reported	Not reported.
Gastro-intestinal tract disturbances.*	Bleeding, rare, diarrhoea uncommon.	Rare	Very rare	Not reported.

† Sulfaguanidine has shown little toxicity to date. Drug rash and fever have been noted.
*Best to stop drug and force fluids.
**Imperative to stop drug and force fluids.

(7) *Laboratory procedures used in following patients receiving sulfonamide compounds.*—(*a*) *General.*—It should be constantly borne in mind that laboratory measures should be utilized as an adjunct in the effort to avoid complications from the administration of sulfonamide compounds. However, in the treatment of *gonorrheal urethritis* in accordance with S. G. O. Circular Letter No. 18, March 10, 1941, the sulfonamide dosage is so small that blood sulfonamide determination and measurement of daily urine output will not ordinarily be required. Careful clinical observation, hemoglobin estimation, and red and white blood counts must be carried out; nevertheless, in all cases, and in those showing any abnormal response to the drug or having a history of such an abnormal response, and in those who have recently taken sulfonamides, or who are suspected of having done so, action of the drug should be carefully followed by appropriate additional laboratory procedures. Hospitalization should be promptly effected in case of complications.

(*b*) *Determination of concentration of sulfonamide compound in blood, body fluids, and urine.*—In patients who are receiving one of the sulfonamide compounds, determination of the concentration of the particular drug in the blood should be made on the morning of the day following the expiration of a full 24-hour period of sulfonamide therapy and should be repeated subsequently when in the opinion of the ward surgeon such a determination will aid in the clinical conduct of therapy. The method of Bratton and Marshall will be employed for the determination of sulfonamide compounds in the blood, body fluids, and urine.

1. Reagents.—The reagents can be obtained through medical supply channels.

(a) A solution of trichloroacetic acid containing 15 grams dissolved in water and diluted to 100 cc.

(b) A 0.1 percent solution of sodium nitrite. Should be prepared fresh each day.

(c) An aqueous solution of N–(1–naphthyl) ethylenediamine dihydrochloride containing 100 milligrams per 100 cc. This solution should be kept in a dark-colored bottle. If kept in ice box when not in use it will keep for one week.

(d) A solution of saponin containing 0.5 gram per liter.

(e) 4 N hydrochloric acid.

(f) A solution of ammonium sulfamate, containing 0.5 gram per 100 cc.

(g) A stock solution of sulfanilamide, sulfapyridine, sulfathiazole, sulfaguanidine, or sulfadiazine in water containing 200 milligrams per liter. The chemically pure, dry, finely powdered drug should be used, *not tablets*. This solution can be kept for several months in the ice box. The most convenient standards to prepare from the stock solution are 1, 0.5, and 0.2 milligrams percent. To prepare these 5, 2.5, and 1 cc of the stock solution plus 18 cc of the 15 percent solution of trichloroacetic acid are diluted to 100 cc. The appropriate standard should be used depending on which drug is being determined in blood.

2. *Procedures for blood.*—Sample and reagent volumes can be proportionately reduced to give the minimal amount of filtrate necessary for an accurate color comparison. Two cc of oxalated blood are measured into a flask and diluted with 30 cc of saponin solution, and after 1 or 2 minutes precipitated with 8 cc of the solution of trichloroacetic acid. The free drug is determined in the filtrate as follows: 1 cc of the sodium nitrite solution is added to 10 cc of the filtrate. After 3 minutes standing, 1 cc of the sulfamate solution is added, and after 2 minutes standing 1 cc of the solution of N–(1–naphthyl) ethylenediamine dihydrochloride is added. The unknown is compared with an appropriate standard which has been treated as above. It is convenient to set the 1 milligram percent standard at mm, the 0.5 at 15 mm, and the 0.2 at 20 mms. This comparison can be made immediately and no change in color is observed for 1 hour or more if solutions are kept in the dark. To determine the total drug, 10 cc of the filtrate are treated with 0.5 cc of 4 N hydrochloric acid, heated in a boiling water bath for 1 hour, cooled, and the volume adjusted to 10 cc. The subsequent procedure is as stated above for determining free drug. The use of a green filter (Corning, Sextant 63,

No. 401, 2 mm thick) facilitates this comparison especially with weak colors. Calculation of results as follows:

$$\frac{\text{Reading of standard}}{\text{Reading of unknown}} \times \text{mg percent in standard} \times 20 = \text{mg percent in blood}$$

(a) For sulfanilamide and sulfaguanidine no corrections are necessary.

(b) For sulfapyridine, no corrections for free or total with less than 5 milligrams percent in blood. When over 5 milligrams percent, multiply both free and total values by 1.1.

(c) For sulfathiazole, multiply free by 1.1 For conjugated, subtract uncorrected value of free from uncorrected value of total and multiply difference by 1.3.

(d) When a photoelectric colorimeter is available, dilutions of blood of 1:50 or 1:100 can be used. The blood is diluted with water (saponin is unnecessary), allowed to stand a few minutes, and precipitated with trichloroacetic acid solution, with a volume which is one-fifth that of the final mixture. This allows the use of 0.1 or 0.2 cc samples of blood which are measured with washout pipettes. Determinations on urine or other body fluids are easily made after appropriate dilution. The reagent blank on distilled water is quite low, but increases with time if the solution is left in the light. For this reason, solutions to be read in the photoelectric colorimeter should be protected from light unless the reading is made immediately. Some reaction occurs between the trichloroacetic acid and the N–(1–naphthyl) ethylenediamine, since solutions acidified with hydrochloric acid do not show an increased color on exposure to light. A calibration curve is made for sulfanilamide and other sul-

fonamide drugs can be read from this curve by using appropriate factors. No standards are necessary after the calibration curve has been determined.

If sulfathiazole or sulfapyridine is being used in the treatment of a patient, or if the patient has decreased kidney function (PSP of 40 percent or below) the determination of sulfonamides should be carried out in a manner which will permit an estimation of the concentrations of both the "free" and "total" drug in the bloods.

(*c*) *Red blood cell and hemoglobin determinations.*—Each patient ((*b*) above) to whom a sulfonamide compound is to be administered should have his hemoglobin determined before sulfonamide therapy is started and once a week thereafter as long as sulfonamide therapy is continued. If in the opinion of the ward surgeon more frequent determinations of the patient's hemoglobin are advisable, these should be done. Red blood counts should be ordered at the discretion of the ward surgeon.

(*d*) *White blood cell counts.*—The total white blood cell and differential count of the patient ((*b*) above) should be determined before therapy with sulfonamide compounds is started. Subsequent cell counts should be done on the 3d, 6th, and 9th days of therapy. If sulfonamide therapy is continued beyond 10 days, these should be repeated every other day thereafter until therapy is discontinued. If, in the opinion of the ward surgeon, sulfonamide therapy is producing a leukopenia of 4,000 W. B. C. or below and this leukopenia is not due to the infection from which the patient is suffering, a second total white blood cell and differential count should be done immediately in order to check the supposedly low count.

(*e*) *Urine examinations.*—The urine output should be measured and recorded daily upon the chart of all patients receiving sulfapyridine, sulfathiazole, or sulfadiazine compounds ((*b*) above). These patients should have their urine examined every 2 days for the presence of red blood cells and albumin. If any degree of macroscopic hematuria develops in the course of sulfonamide therapy the drug should always be stopped. The appearance of microscopic hematuria does not necessarily contraindicate a continuance of chemotherapy. When present the urine output should be carefully followed and daily urine examination performed. If the urine output falls noticeably the drug should

be stopped. The finding of sulfonamide drug crystals in the urine of patients receiving these drugs does not in itself constitute an indication for stopping the drug.

(*f*) *Bacterial studies.*—An attempt should be made in every patient in whom sulfonamide therapy is contemplated to establish the etiological diagnosis of the infection by bacteriological methods. However, this does not mean that sulfonamide treatment should be delayed until the bacteriological diagnosis is established. If the clinical diagnosis is that of an infection known to respond to sulfonamide therapy, then the appropriate drug should be used at once. All bacteriological culture media used in the studies of patients who are receiving sulfonamide compounds should contain 2.0 milligrams percent of para aminobenzoic acid. This chemical neutralizes the bacteriostatic effect of the sulfonamides in culture media. If para aminobenzoic acid is not available a similar concentration of procaine hydrochloride may be used.

d. Serum should never be given to patients who have a history of asthma, or those who have a history of sensitivity to animal dander or serum. *Whenever serum is administered*, have a syringe containing 1 cc of epinephrin 1 : 1,000 solution at the bedside of the patient in order that this drug may be injected rapidly if an anaphylactic reaction develops. Two tests for serum sensitivity are available and it is desirable to use both. The first consists of the intracutaneous injection of 0.1 cc of a solution of the serum diluted 1 to 10 in sterile physiological saline. The test is positive if at the end of 20 minutes the wheal of the injection has enlarged to the size of a nickel and has an area of redness about it. The second test is the conjunctival test. This test consists of instilling one drop of a 1 to 10 dilution of the serum in sterile physiological saline inside the lower eyelid of the patient. If redness and swelling of that eye appear within 20 minutes, the test is positive. A positive reaction can be counteracted easily by instilling one or two drops of a 1 to 1,000 solution of epinephrin inside the lower eyelid, a procedure which should be followed in all positive reactors in order to prevent rare instances of corneal ulceration. These tests are interpreted as follows:

(1) If the eye and skin tests are negative, then antitoxin may be given by any route.

(2) A definite wheal-like reaction with surrounding erythema indicates skin sensitivity.

(3) A positive ophthalmic reaction with or without a positive intradermal test indicates a probable systemic reaction. Serum should never be given if the ophthalmic and skin tests are positive, and only with the greatest of care if either the ophthalmic or skin test is positive. This warning also applies to serum treatment of meningococcal meningitis, pneumococcal pneumonia, and gas-bacillus infections.

43. Hemolytic streptococcus infections.—*a. Mild or moderately severe hemolytic streptococcus infections, such as erysipelas, mild cellulitis (including wound infections), and tonsilitis.*—(1) Proper surgical treatment should be given, if necessary; this includes the opening of all wounds, for the full length of the infected portion. All localized spontaneous or metastatic foci of infection should be drained, but not until at least 2 hours after the starting of chemotherapy.

(2) Immobilization, elevation, and hot, wet dressings favor the localization and resolution of wound infections.

(3) Sulfadiazine is recommended as the drug of choice, given as follows:

(*a*) *Initial dose (oral).*—4.0 grams (60 grains), due allowance being made for previous administration.

(*b*) *Subsequent doses.*—1.0 gram (15 grains) every 4 hours day and night until 5 days of normal temperature have elapsed. If the blood concentration of sulfadiazine exceeds 10 milligrams percent this dose may be decreased accordingly. The subsequent dosage may be modified after 48 hours of normal temperature at the discretion of the medical officer.

b. Otitis media.—This condition is generally caused by hemolytic streptococci, but may be caused by pneumococci or other organisms and present a special case. Bacteriological cultures should be taken. The treatment should be started as outlined in *a* above and continued according to the organism found on culture.

(1) Treatment should be given as outlined above; sulfadiazine should be continued in doses of 0.5 gram (7½ grains) every 6 hours for at least 10 days after a *clinical* cure has been effected.

(2) A blood concentration of 5 to 10 milligrams percent (free drug)[5] of sulfadiazine is desirable in mild and moderately severe hemolytic streptococcal infection.

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[5] References to blood concentration throughout this section refer to "free" drug.

c. Severe hemolytic streptococcus infections, such as meningitis, septicemia, severe cellulitis, acute osteomyelitis, and acute mastoiditis.—(1) Proper surgical and supportive measures should be provided (see above).

(2) Orally administered sulfadiazine is recommended as the drug of choice.

(*a*) *Initial dose (oral).*—4.0 grams (60 grains).

(*b*) *Subsequent doses.*—These should be 1 gram (15 grains) every 4 hours day and night until temperature has been normal for 7 days.

(3) In acute streptococcal mastoiditis or osteomyelitis sulfadiazine should be continued in small doses of 0.5 gram (7½ grains) four times a day for at least 10 days after a clinical cure has been effected.

(4) Sodium sulfadiazine, intravenously, may be used in severe hemolytic streptococcal infections. (See par. 42*c*(5).) If it is used, the initial dose is 0.10 gram per kilo of body weight. Following the initial intravenous dose of sodium sulfadiazine it is generally sufficient to continue therapy with 1.0 gram of sulfadiazine by mouth every 4 hours day and night until the temperature has been normal for 7 days. If, following the initial intravenous dose of sodium sulfadiazine, it is desired to continue therapy by the intravenous rather than the oral route, subsequent doses of sodium sulfadiazine based on 0.05 gram per kilo of body weight, administered at 12-hour intervals by the intravenous route, should be used. It is always advisable to begin oral therapy with sulfadiazine as soon as possible.

(5) A blood concentration of sulfadiazine of 15 milligrams percent (free drug) should be maintained during the febrile phase of serious hemolytic streptococcal infections.

44. **Scarlet fever.**—*a. Active immunization with toxin.*—(1) This is not generally recommended for the following reasons:

(*a*) A high percentage of adults are immune.

(*b*) Five or more injections of toxin are usually required to produce immunity.

(2) Active immunization should be used in Dick positive nurses and orderlies assigned to the care of scarlet fever patients.

b. Simple toxic scarlet fever (exanthematous stage).—(1) Antitoxin (globulin concentrated) is recommended for all moderately severe to extremely severe cases in patients who do not have a history of asthma and who are not hypersensitive to the

serum (pars. 22 and 42); it should be given in one dose as soon as the diagnosis is made, intramuscularly in moderately severe cases and intravenously in severe to extremely severe cases according to the following schedule of dosage:

(*a*) Moderately severe, 18,000 units.

(*b*) Severe, 27,000–36,000 units.

(*c*) Very severe, 45,000 units.

(2) Sulfadiazine exerts no therapeutic effect on the toxic stage; it should be used for prophylaxis of septic complications, giving 0.5 gram (7½ grains) every 6 hours for the period of quarantine.

c. Toxic and septic scarlet fever (exanthematous stage).—(1) Antitoxin (globulin concentrated) is recommended. Cases with early septic lesions (purulent rhinopharyngitis, sinusitis, otitis media, mastoiditis, marked lymphadenitis, etc.), in general, are actually or potentially more toxic than simple toxic (uncomplicated) cases. The dose should be larger, according to the following schedule:

(*a*) Moderately severe, 27,000 units.

(*b*) Severe, 45,000 units.

(*c*) Very severe, 63,000 units.

(2) Sulfadiazine should also be given because of its chemotherapeutic effect on septic lesions; the schedule of dosage is as follows:

(*a*) *Initial dose (oral).*—4.0 grams (60 grains).

(*b*) *Subsequent doses.*—1.0 gram (15 grains) every 4 hours day and night until temperature has been normal for 5 days; then 0.5 gram (7½ grains) every 6 hours for the period of quarantine.

d. Late septic scarlatinal complications (post-exanthematous stage).—(1) Antitoxin is of no value.

(2) Sulfadiazine is recommended, with the dosage and duration of treatment as in the preceding section.

45. Meningococcal meningitis.—*a. Serotherapy.*—It is generally recommended that antimeningococcus serum should not be used in the treatment of meningococcal meningitis. However, in individual cases, serum may be used if, in the opinion of the medical officer, it is indicated and if the patient is not hypersensitive to the serum (par. 42).

b. Chemotherapy.—(1) Sulfadiazine is recommended as the drug of choice. It should *not* be injected intrathecally. (If not

available, give sulfanilamide in the same doses by mouth.) Sulfadiazine is given as follows:

(*a*) *Initial dose* (*oral*).—4.0 grams (60 grains).

(*b*) *Subsequent doses.*—1.0 gram (15 grains) every 4 hours day and night until the temperature has been normal for 7 days.

(2) Sodium sulfadiazine is given as follows:

(*a*) *Initial dose* (*intravenously only*).—0.10 gram per kilo of body weight.

(*b*) *Subsequent doses.*—0.05 gram per kilo of body weight at 12-hour intervals.

(3) A blood concentration of sulfadiazine of 15 milligrams percent should be maintained during the febrile period of the disease.

(4) It is always advisable to begin oral therapy with sulfadiazine as soon as possible.

c. Lumbar puncture.—This is indicated for diagnosis, and subsequently only to relieve manifestations of increased intracranial pressure.

46. Other forms of purulent meningitis (pneumococcic, staphylococcic, and cases of unknown origin).—If the etiology of a purulent meningitis is not promptly established, chemotherapy should, nevertheless, be instituted at once; sulfadiazine is the drug of choice, given as follows:

a. Orally.—(1) *Initial dose.*—4.0 grams (60 grains).

(2) *Subsequent doses.*—1.0 gram every 4 hours day and night until the temperature has been normal for 7 days. A blood concentration of at least 10 to 15 milligrams per 100 cc is desirable, and where these determinations can be obtained, enough drug should be given to establish and maintain approximately this concentration until there is an apparent cure of the disease.

b. Intravenously (if oral treatment is impossible).—(1) *Initial dose.*—0.10 gram of sodium sulfadiazine per kilogram of body weight.

(2) *Subsequent doses.*—Should be based on 0.05 gram per kilogram of body weight and should be given at 12-hour intervals; oral therapy with sulfadiazine should be started as soon as it is practicable.

47. Pneumonia.—*a. General.*—(1) The specific treatment of pneumonia is based on etiologic classification and diagnosis, which should be carried out promptly in all cases whenever laboratory diagnostic facilities are available.

(2) Pneumonia is classified in two main groups:

(*a*) *Primary*—occurring independently of any major predisposing cause.

(*b*) *Secondary*—occurring as a complication of a major predisposing cause, for example, influenza, measles, or pertussis.

(3) Primary pneumonia is principally caused by the pneumococcus; other bacteria, for example, beta-hemolytic streptococcus, or Friedlander's bacillus, may occasionally be responsible.

(4) Secondary pneumonia complicating influenza, measles, and other diseases are of variable etiology, often mixed, sometimes indeterminate. The higher types of pneumococci, beta-hemolytic streptococci, and the influenza-bacillus are the most frequent bacteria concerned; straphylococci, gram-negative micrococci, and alpha-hemolytic strepococci may be found.

b. Primary pneumonia.—(1) *Pneumococcal pneumonia (usually lobar).*—(*a*) Chemotherapy is recommended as the method of choice in all cases for the following reasons:

1. It is highly effective, except in the aged with chronic disease.

2. It is effective against all pneumococcal types.

3. It may be given at once without waiting for type determination.

4. Its administration is technically simple and relatively inexpensive.

5. It is also applicable to most bacterial pneumonias other than pneumococcal.

(*b*) Sulfadiazine is recommended as the drug of choice for all cases for the following reasons:

1. It is greatly superior to sulfanilamide in scope and effectiveness of therapeutic action.

2. It is at least equivalent to sulfathiazole and to sulfapyridine in scope and effectiveness of therapeutic action.

3. It causes much less nausea, vomiting, and mental disturbance than does sulfathiazole or sulfapyridine.

4. Other untoward reactions are less frequent and severe than they are with sulfathiazole or sulfapyridine.

5. The problem of excessive acetylation is not encountered to the same degree as it is with sulfathiazole or sulfapyridine. (If sulfadiazine is not available use sulfathiazole in the same doses by mouth.)

(*c*) Method of treatment in chemotherapy.

1. Sulfadiazine should be started at once in all cases as soon as the clinical diagnosis is made, an attempt at etiologic diagnosis being carried out at the same time.

2. The initial dose (oral) should be 4.0 grams (60 grains), and the subsequent doses, 1.0 gram (15 grains) every 4 hours day and night until 72 hours of normal temperature have elapsed.

3. In very severe cases an initial dose of 0.10 gram per kilo of body weight of a 5 percent solution of sodium sulfadiazine may be given intravenously.

4. In patients unable to take sulfadiazine by mouth, intravenous treatment with a 5 percent solution of sodium sulfadiazine is also recommended; the initial dose should be 0.10 gram per kilo of body weight of the drug, and the subsequent doses 0.05 gram per kilo of body weight repeated every 12 hours; change to oral dosage as soon as possible.

5. Force fluids to 3,000–4,000 cc per day.

(*d*) Serotherapy, that is, the injection of homologous-type antipneumococcus serum, preferably rabbit serum, is recommended in addition to sulfadiazine therapy in early cases (less than 72 hours after onset) only when the patients have failed to show satisfactory response to 48 hours of chemotherapy (this happens very rarely) and in late cases (more than 72 hours after onset) that clinically appear unusually severe and are presumptively or known to be bacteremic or that have failed to respond to chemotherapy alone.

(*e*) Method of treatment in serotherapy:

1. If the patient is not hypersensitive to serum (par. 42), it is recommended that an initial dose of 200,000 units of antipneumococcus rabbit serum of homologous type be given intravenously in early cases that have not responded to chemotherapy.

2. An initial dose of 300,000 units should be given in late, severe, presumptively bacteremic cases.

3. If these prove insufficient, subsequent doses of 100,000 units each should be given every 8 hours.

4. Force fluids to 3,500 cc per day.

(2) *Hemolytic streptococcus pneumonia, Friedlander's bacillus pneumonia.*—(*a*) Sulfadiazine is recommended in all cases; the

schedule of dosage is as follows:

1. *Initial dose (oral).*—4.0 grams (60 grains).
2. *Subsequent doses.*—Begin with 1.0 gram (15 grains) every 4 hours; if response is not satisfactory increase to 1.5 grams (22½ grains) every 4 hours until improvement is definite, then reduce dose to 1.0 gram (15 grains) every 4 hours and continue until the temperature has been normal for 5 days.

(*b*) Sodium sulfadiazine, if indicated, should be administered intravenously as in pneumococcal pneumonia (see above).

c. Staphylococcal pneumonia.—(1) Sulfathiazole is recommended in all cases.

(*a*) *Initial dose (oral).*—4.0 grams (60 grains).

(*b*) *Subsequent doses (oral).*—Begin with 1.0 gram (15 grains) every 4 hours; if response is not satisfactory increase to 1.5–2.0 grams (22.5–30 grains) every 4 hours until improvement is definite, then reduce dose to 1.0 gram (15 grains) every 4 hours and continue until temperature has been normal for 5 days.

(2) Sodium sulfathiazole is given by the intravenous route as a 5 percent solution. It is advisable to change to oral dosage as soon as possible.

(*a*) *Initial dose.*—4.0 grams (60 grains).

(*b*) *Subsequent doses.*—2.0 grams (30 grains) every 6 hours.

d. Secondary pneumonia.—(1) *Prophylaxis.*—The possible value of chemotherapy with sulfonamide compounds in the prevention of bacterial pneumonias complicating influenza, measles, and other diseases is not known at present; specific prophylactic recommedations are therefore not justified.

(2) *Chemotherapy.*—Sulfadiazine is recommended in all cases in which pneumococci, hemolytic streptococci, or Friedlander's bacilli are found and believed to be of etiologic significance. Sulfathiazole is recommended in cases in which staphylococci predominate. The method of treatment, dosage, and precautions are the same as in primary pneumonia (see above).

(3) *Comment.*—Chemotherapy with sulfonamide derivatives is of no demonstrable value in many bronchopneumonias of indeterminate (virus ?) etiology or in pulmonary infections due to alpha-hemolytic streptococci and *Haemophilus influenzae;* results in secondary pneumonias, which are often mixed infections, are therefore variable, often disappointing, and difficult to evaluate,

even when bacteria known to be susceptible to the sulfonamide compounds are present in the sputum.

48. Gas-bacillus infection.—*a. General.*—The problem of gas-bacillus infection is largely one of prevention, and all wounds in which such infection is a possibility should receive proper surgical treatment (par. 3) at the earliest possible moment.

b. Prophylaxis.—Sulfanilamide is recommended as the drug of choice, the initial dose being 6.0 grams (90 grains) oral and subsequent doses 1.0 gram (15 grains) every 4 hours day and night. This should be continued for 7 days or until definitive treatment is available. This period of therapy almost always eliminates the possibility of gas-bacillus infection. Crystalline sulfanilamide should be used *locally.* It should be distributed evenly over the surface of the wound, approximately .01 gram (1½ grains) being used per square inch but not over 10 grams (150 grains) for any one person.

c. Treatment.—(1) *General.*—The primary wound should be opened, and *all* infected tissue should be removed (in occasional cases, this may be so extensive as to warrant amputation).

(2) *Chemotherapy.*—(*a*) Sulfathiazole is recommended at present as the drug of choice.

(*b*) The dosage is as follows:

1. Initial dose (oral).—6.0 grams (90 grains).

2. Subsequent doses (oral).—1.0 gram (15 grains) every 4 hours day and night until the temperature has been normal for 48 hours; then 0.5 gram (7½ grains) every 4 hours day and night until convalescence is completely established.

(3) *Serum therapy.*—Polyvalent gas gangrene antitoxin should be used when in the opinion of the medical officer it is indicated. It should be administered in adequate dosage according to the directions inclosed in each individual package.

(4) *Local chemotherapy.*—(*a*) If all grossly infected tissue cannot be surgically removed, sulfathiazole powder should be applied locally, being used in the same way as sulfanilamide powder (*b* above).

(*b*) If all grossly infected tissue appears to have been removed, a paste of zinc peroxide may be applied. This is made by mixing a medicinal grade of zinc peroxide with an approximately equal amount of sterile distilled water or physiologic saline solution, to form a smooth, creamy suspension, which

flows readily to all parts of the wound. The wound is then covered with a thick layer of cotton, wet with water or saline solution, over which is placed a layer of rubber, cellophane, or vaseline gauze, to prevent evaporation. A fresh dressing should be applied every 1 or 2 days, washing out the exudate and old zinc peroxide with sterile physiologic saline solution, and these should be continued until the infection has been controlled.

(5) *Adequate fluids* (3,000–4,000 cc daily) should be provided.

49. Tetanus.—*a. General.*—As in gas-bacillus infection, the problem of tetanus is largely one of prevention, and all wounds should receive proper surgical treatment as soon as possible.

b. Prophylaxis.—(1) The proper prophylactic procedure depends on whether it is definitely known that the patient has been immunized with a *full course* of injections of tetanus toxoid, the "initial vaccination," with or without "subsequent vaccinations," as specified in Circular Letter Number 34, Office of The Surgeon General, April 16, 1941.

(2) In the presence of known and adequate immunization, an emergency "stimulating" dose (1 cc) of tetanus toxoid should be given subcutaneously to the following:

(*a*) All persons who incur wounds or severe burns on the battlefield.

(*b*) Patients undergoing secondary operations or manipulations of old wounds, if toxoid has not been given within the preceding 3 months.

(*c*) Those who incur punctured or lacerated nonbattle wounds, powder burns, or minor injuries that might be complicated by the introduction of *Clostridium tetani* into the tissues.

(3) In the absence of previous immunization or if immunization was incomplete or questionably complete, a passively immunizing dose (1,500 units) of tetanus antitoxin should be injected subcutaneously, due care being taken that the patient is not sensitive to the serum (par. 42); in addition, all such patients should be vaccinated with tetanus toxoid, receiving the specified course for "initial vaccination" (see above), that is, three subcutaneous injections of 1.0 cc of tetanus toxoid at 3-week intervals.

c. Treatment.—(1) At the appearance of the earliest signs of tetanus, immediate therapy is indicated; all cases must be treated vigorously.

(2) The patient should be secluded, if possible, in a quiet, darkened room.

(3) Spasms must be controlled by heavy doses of either paraldehyde or chloral hydrate. Patients with tetanus vary widely in their response to these drugs and each case must be treated individually. From 8 to 40 cc of paraldehyde can be given by rectum, and this dose can be repeated every 4 hours. Chloral hydrate can be given in doses of from 1 to 3 grams, either by mouth or rectum; this dose can be repeated every 4 hours or more often if necessary to control spasms. In severe cases, 0.03 to 0.06 gram (½ to 1 grain) avertin per kilogram of body weight should be given by rectum. With all these sedatives, care must be exercised to avoid marked respiratory depression. This heavy sedation is continued during the course of the severe phase of tetanus. If respirations cease, it is imperative to use artificial respiration (par. 29).

(4) The primary wound should be treated as follows: the area around the injury is infiltrated with from 5,000 to 10,000 units of tetanus antitoxin, due precautions being taken against serum sensitivity (par. 42). An hour later the wound is incised and left open; foreign bodies must be removed and adequate debridement carried out, when indicated.

(5) Tetanus antitoxin must be administered early and in adequate amounts. If the patient is sensitive to serum (par. 42), desensitization should be carried out, when feasible. A syringe containing a 1:1000 solution of epinephrine (adrenalin) should always be at hand. The schedule of dosage for antitoxin is as follows: 60,000 units is given intravenously and 40,000 units intraspinally (in severe cases the intravenous dose is repeated in 24 hours); on the second day and every day thereafter until definite improvement occurs, approximately 5,000 units is given intravenously. The intravenous and intraspinal injections must be given *very* slowly. If anaphylactic symptoms develop during treatment, immediate withdrawal of the needle is imperative and frequent doses (0.5 to 1.0 cc) of a 1:1000 solution of epinephrine (adrenalin) should be given.

(6) Tracheotomy should be performed if laryngeal spasm is causing suffocation.

(7) A liberal fluid diet (2,000 to 4,000 calories) should be given, and the patient must be kept in fluid balance.

(8) Constant nursing care should be provided, if possible.

50. **Staphylococcal infections.**—*a. Localized infections.*—(1) Apply hot, wet dressings until definite fluctuation develops.

(2) Lesions that do not evacuate themselves spontaneously after becoming fluctuant should be promptly incised and drained.

(3) Chemotherapy is not recommended for minor staphylococcal infections.

(4) Patients with facial infections may develop serious complications and should be handled with conservatism; chemotherapy may be indicated (see below).

b. Large boils, carbuncles, and generalized infections.—(1) Apply hot, wet dressings, and incise or excise (carbuncles) as soon as fluctuation develops; the incision of uninfected tissue should be avoided.

(2) Sulfathiazole is recommended as the drug of choice in serious staphylococcal infections and should be given to any patient in whom one or more of the following factors is present:

(*a*) A white cell count of 12,000 or above.
(*b*) A temperature of 101° F. or above.
(*c*) A chill.
(*d*) A palpably thrombosed vein.
(*e*) A pulmonary infarct.
(*f*) A positive blood culture.

(3) The initial oral dose of sulfathiazole should be 4 grams (60 grains), followed by 1.0 gram (15 grains) every 4 hours until the temperature has been normal for 48 hours, then 1 gram (15 grains) every 4 hours for 14 days or longer.

(4) Patients unable to take sulfathiazole by mouth should receive sodium sulfathiazole intravenously.

(5) Sulfathiazole powder may also be applied locally, being used in the same way as sulfanilamide powder (par. 48).

(6) The fluid intake should total at least 3,000 cc daily, and the urinary output should be carefully recorded, to safeguard against renal shutdown.

(7) Employ surgical treatment for any areas of localized suppuration that may develop. Metastatic suppurative foci usually indicate the existence of a septic thrombophlebitis; such foci should be immediately drained, and the thrombosed vein ligated, if possible.

(8) Persistent illness after presumably adequate drainage indicates the necessity for a thorough search for unrecognized foci of infection.

c. Staphylococcal bacteremia.—(1) Give sulfathiazole.

(*a*) *Initial dose* (*oral*).—4.0 grams (60 grains).

(*b*) *Subsequent doses* (*oral*).—1.5 grams (22½ grains) every 4 hours until the temperature has been normal for 48 hours; then reduce the dose to 1.0 gram (15 grains) every 4 hours and continue at this level for 14 days. The likelihood of relapse is very great unless prolonged, continuous chemotherapy is employed.

(2) Make every effort, through careful clinical and X-ray examinations, to identify foci of localized infection; these should be treated by surgical drainage if they are accessible. Sulfathiazole may control invasive manifestations of infection but will not, by itself, bring about a cure of areas of localized infection; reinvasion from such areas is likely, if they are not drained.

d. Chronic staphylococcal suppuration (such as chronic osteomyelitis).—(1) Maintain drainage by packing with vaseline gauze to the depths of the wound until sequestration of necrotic bone and tissue has occurred and the wound is covered with clean granulations.

(2) The oral administration of sulfathiazole may be of some value.

(3) The local application of powdered sulfathiazole is of value.

51. Wound infections.—*a. General.*—The problem of wound infection is largely one of prophylaxis, which includes surgical and supportive care and the administration of certain specific agents. When infection has developed, definite treatment is indicated.

b. Prophylaxis.—(1) Proper surgical and supportive care should be given (sec. II).

(2) Polyvalent gas-bacillus antitoxin should be given, if indicated (par. 48).

(3) Tetanus toxoid or antitoxin, or both, should be given if indicated (par. 49).

(4) Chemotherapy should be immediately instituted.

(*a*) Give 2.0 grams (30 grains) sulfadiazine by mouth, and then 1 gram (15 grains) every 4 hours, day and night, for 7 days or until definitive treatment is indicated. The amount of a sulfonamide already taken by the injured man should be ascertained and the subsequent dosage reduced accordingly; proper consideration to dosage should also be given to patients with

urinary suppression due to hemorrhage, shock, or dehydration. If adequate amounts of fluid (2,500 to 3,000 cc) cannot be given, the dosage also should be lowered, to prevent an abnormally high concentration of the drug.

(*b*) In cases in which sulfadiazine cannot be given by mouth it can be given intraveneously (par. 43).

(*c*) Sulfanilamide powder should be lightly dusted on the wound.

c. Treatment.—If infection develops, definitive treatment according to the recommendations outlined in other paragraphs in this section should be given. Infections due to certain miscellaneous anaerobic organisms (nonhemolytic streptococci, nonsporulating bacilli, fusiform bacilli, and spirochetes), although not so potentially dangerous as tetanus and gas-bacillus infection, demand special local chemotherapy (par. 48).

52. Peritonitis (secondary to appendicitis or bowel perforation).—In addition to the surgical procedures indicated in dealing with peritonitis secondary to appendicitis or bowel perforation, the following is recommended regarding chemotherapy:

a. Local treatment.—Sulfanilamide crystals, in an amount between 4.0 and 8.0 grams (60–120 grains) should be applied to the peritoneum in the immediate area exposed at operation, and to the layers of the abdominal wound, during its closure.

b. Systemic treatment.—Parenteral sulfanilamide, 150 cc of 1 percent solution (1.5 grams), should be given every 6 hours by hypodermoclysis, starting before or immediately after operation. Continue this dosage for 2 days. If the patient shows satisfactory progress at that time the dose may be reduced to 120 cc (1.2 grams) every 6 hours. As soon as the patient is allowed oral feedings the sulfanilamide may be given by mouth in a dose of 1.0 gram (15 grains) every 4 hours. It is usually unnecessary to continue treatment beyond the 6th post-operative day. If no sulfanilamide is implanted locally, the initial dose of the solution by hypodermoclysis should be 400 cc. (See par. 42*c*(5)(*b*).)

53. Urinary tract infections.—*a. General considerations.*—It has been definitely shown that obstruction due either to renal or bladder calculi or any other type of pathological obstruction of the urinary tract greatly militates against the successful use of sulfonamide and other compounds in the treatment of urinary tract infections. Hence, when such pathological conditions are present, good results in the treatment of urinary

tract infections are usually not obtained and every effort should be made to eradicate the obstruction. It is advisable in the therapy of urinary tract infections to adjust the fluid intake of the patient so that his *urine output is in the neighborhood of 1,000 cc per day.* If signs of renal impairment (lowered PSP) are present, doses of the drug should be decreased and the concentration of the drug in the blood determined daily in order that concentrations of more than 10 milligrams percent of the drug in the blood be avoided. Bacteriological cultures designed to determine the sterility of the urine should always be employed, and it is desirable to include in the bacteriological procedures a culture made by placing 5 cc of urine on a blood agar slant containing 2 milligrams percent of para aminobenzoic acid, this to be incubated for 5 days before being discarded as sterile.

b. (1) *Infections due to E. coli, A. Aerogenes, Shigella dispar and other gram negative organisms belonging to the so-called typhoid-paratyphoid group.*

(2) *Specific treatment.*—Sulfadiazine is the drug of choice. Dosage, oral: 1.0 gram (15 grains) every 4 hours day and night until definite clinical improvement has been noted. Then decrease dose to 1.0 gram (15 grains) 4 times a day until the urine is clear and 2 negative cultures have been obtained in medium containing para aminobenzoic acid. Then stop drug, wait one week, and reculture the urine. If sulfadiazine is not available, sulfathiazole is the second drug of choice, sulfanilamide the third. The dosage is the same for the latter two drugs.

c. (1) *Infections due to staphylococcus aureus.*

(2) *Specific treatment.*—Sulfathiazole is the drug of choice. Dosage, oral: 1.0 gram (15 grains) every 4 hours day and night until definite clinical improvement has occurred. Then, decrease dose to 1.0 gram (15 grains) 4 times a day and maintain this until 2 negative urine cultures have been obtained in medium containing para aminobenzoic acid. Then stop the drug and reculture the urine one week later.

d. (1) *Infections due to Pseudomonas aeruginosa (B. pyocyaneus).*

(2) *Specific treatment.*—Sulfathiazole is the drug of choice. Dosage, oral: same as for staphylococcal urinary tract infections.

e. Infections due to B. proteus.—Sulfathiazole is the drug of

choice. Dosage, oral: same as for staphylococcal urinary tract infections.

f. (1) *Infections due to entercoccal organisms* (*Streptococcus fecalis*).—(These infections do not respond to sulfonamide drugs, and hence, such drugs are not used.)

(2) *Specific treatment.*—Ammonium mandelate is the drug of choice. Dosage, oral: 3 grams (45 grains) 4 times a day for a period of 5 to 7 days. Sufficient drug should be administered to keep the pH of the urine at 5.0 or below. If violent nausea or vomiting appears the drug should be stopped.

g. Miscellaneous urinary tract infections.—If organisms other than those already described are the etiological agent of the urinary tract infection, either sulfadiazine or sulfathiazole should be used in doses advised above.

Section VI

TREATMENT AND CONTROL OF CERTAIN TROPICAL DISEASES

54. General.—The following notes on the treatment and prevention of certain tropical diseases among military personnel are based on recommendations prepared at the request of The Surgeon General by the Subcommittee on Tropical Diseases, and approved by the Committee on Chemotherapy and the Committee on Medicine of the Division of Medical Sciences, National Research Council, as published in Circular Letter Number 56,

Office of the Surgeon General, June 9, 1941. The information is offered only as a general guide in the handling of tropical diseases and is for use at the discretion of the medical officer. It is not intended as a substitute for the more comprehensive publications available on the subject.

55. Amebic dysentery and amebiasis.—*a. Etiologic agent.*—*Endamoeba histolytica.*

b. Geographic distribution.—The distribution of this disease is probably world-wide, but infections are much more common in the tropics and subtropics than in temperate regions. Even in the latter regions the incidence may be high in localities where sanitary conditions are poor.

c. Transmission.—The disease is transmitted through the ingestion of food or drink contaminated with feces of carriers or cases containing the cysts, or possibly the trophozoites of *E. histolytica.*

d. Specific diagnosis.—The diagnosis of *E. histolytica* infection depends on the demonstration of the parasite in the feces or tissues of the infected individual. The complement-fixation test is a valuable aid to diagnosis but should be supported where possible by microscopic demonstration of the parasite. Cultivation methods are useful in diagnosis.

e. Treatment.—(1) *Ameba carriers without dysentery.*—Give either carbarsone or chiniofon as follows:

(*a*) Carbarsone: 0.25 gram (3¾ grains) by mouth twice a day for 10 days; give a soft diet, if possible, and restrict the activities of the patient. Toxic symptoms (rare) consist of abdominal distress, nausea, vomiting, or exfoliative dermatitis.

(*b*) Chiniofon: 1 gram (15 grains) by mouth three times a day for 10 days. Give a soft diet and bed rest, if possible. Watery diarrhea is evidence of toxicity.

(*c*) Beginning 7 days after completion of treatment examine the stools for cysts on 3 consecutive days. Make similar examinations once a week for 4 weeks thereafter and reexamine 3 months subsequently on 3 consecutive days. Repeat the treatment if cysts are still present.

(2) *Acute or chronic amebic dysentery.*—(*a*) Inject 0.03 gram (½ grain) emetine hydrochloride intramuscularly twice a day or 0.06 gram (1 grain) once a day for 4 to 6 days. The toxic effects include vomiting (controllable by sedatives), acute myocardial degeneration, and peripheral neuritis (from prolonged

treatment). The injections of emetine should be stopped as soon as the dysenteric symtoms have subsided.

(*b*) Simultaneously with the beginning of the use of emetine, give carbarsone or chiniofon by mouth, as outlined above, but if the treatment is ineffective because of lesions in lower colon or rectum, give carbarsone or chiniofon by rectum as follows:

1. Dissolve 2.0 grams (30 grains) carbarsone in 200 cc of a 2 percent solution of sodium bicarbonate (carbarsone is insoluble in water) and give this solution as a retention enema following a cleansing enema of 2 percent solution of sodium bicarbonate (retention may be aided by a mild sedative); repeat on the 4 following nights, but if it proves irritating give on alternate nights.
2. Dissolve 3.0 grams (45 grains) chiniofon in 300 cc of sterile water and administer the solution similarly to carbarsone, using a cleansing enema of water.

(*c*) For acute cases, keep the patient in bed on a liquid and soft diet (broth at first, later add milk, eggs, custard, and other bland foods) until the acute symptoms subside, then give a soft, low-residue diet. For chronic cases, keep patient in bed on a soft, low-residue diet, and resume a full general diet gradually during convalescence.

(3) *Amebic hepatitis, without liver abscess.*—(*a*) Give emetine hydrochloride, as above, but continue for 8 days.

(*b*) If evidence of hepatitis persists after 8 days, suspect liver abscess and consider aspiration (see below).

(*c*) If practicable, check the effect of emetine on the heart muscle by electrocardiograms before treatment and daily from 5th day on (look for changes in the QRS complex and inversion of T-waves.

(*d*) Use chiniofon, as above, by mouth for intestinal infection, which probably exists (carbarsone may be toxic in some cases of hepatitis).

(4) *Amebic liver abscess.*—(*a*) Use emetine hydrochloride, as above.

(*b*) After 2 to 4 days of emetine treatment drain the abscess, preferably by aspiration; repeat, if necessary, or drain continuously through the aspiration wound (open drainage should be avoided if possible).

(*c*) Use chiniofon, as above, for intestinal infection, which probably exists.

f. Prevention.—The prevention of *E. histolytica* infection depends on measures that will prevent the ingestion of food or drink contaminated with this parasite.

56. Bacillary dysentery.—*a. Etiologic agents.*—The dysentery bacilli (genus Shigella).

b. Geographic distribution.—Dysentery exists throughout the world, especially in unsanitary localities. Its incidence is highest in tropical and subtropical regions.

c. Transmission.—The disease is transmitted through the ingestion of food or drink contaminated with the feces of cases or carriers of dysentery bacilli.

d. Specific diagnosis.—Typical cases present characteristic symptoms, including fever, cramps, diarrhea, and tenesmus, and the feces contain *pus*, mucus, and blood. Specific diagnosis depends on the identification of the etiologic agent in stool cultures.

e. Treatment.—(1) *Chemotherapy.*—Sulfaguanidine is the drug of choice.

(*a*) In patients ill with *acute* bacillary dysentery, initial dose is 3.5 grams (52½ grains); maintenance dose 3.5 grams (52½ grains) every 4 hours day and night, until the number of stools per day is reduced to 5 or less. Then shift to a maintenance dose of 3.5 grams (52½ grains) every 8 hours, day and night, and continue until the stools have been normal for 96 hours.

(*b*) In patients ill with *chronic* bacillary dysentery, 3.5 grams (52½ grains) of sulfaguanidine every 8 hours, day and night. The duration of treatment should not exceed two weeks.

(2) *Supportive treatment.*—(*a*) Complete rest in bed.

(*b*) For mild and moderately severe cases without dehydration or severe toxemia the fluid intake should be 3,000 cc or more every 24 hours.

(*c*) For acute fulminant cases give intravenous glucose, 50 grams in physiological salt solution 1,000 cc sufficient to maintain a daily urinary output of 1,000 cc or more. Give also thiamin hydrochloride 2 milligrams, ascorbic acid 50 milligrams.

(3) *Serum therapy.*—*Shiga antiserum* or *polyvalent antiserum* advised only for fulminating and *markedly toxic* cases; 40–80 cc depending on potency, administered intravenously twice daily until temperature falls to normal. Test the patient for sensi-

tization before administering serum (par. 42). The use of antiserum should not preclude the adequate employment of sulfaguanidine ((1) above).

(4) *Diet.*—Fluid, or free from residue.

f. Prevention.—Use sanitary measures similar to those employed for the prevention of other enteric infections.

57. Cholera.—*a. Etiologic agent.*—*Vibrio comma.*

b. Geographic distribution.—The disease is endemic in Asia, but since ancient times, many pandemics have originated in endemic centers in India and have spread over the world.

c. Transmission.—The disease is transmitted through the ingestion of food or drink contaminated with feces containing *V. comma.*

d. Specific diagnosis.—By the identification of *V. comma* in cultures of feces.

e. Treatment.—(1) Keep the patient in bed and apply heat to the abdomen and extremities as long as required. Watch the blood pressure; if it drops below 100 systolic, give saline or serum (see below).

(2) When the patient can tolerate food, the diet should be low in residue and supplemented with an adequate vitamin intake.

(3) Oral medication includes the following:

(*a*) Give two 0.13 gram (2 grains) potassium permanganate or calcium permanganate pills, enteric coated, every 30 minutes, until the stools turn green.

(*b*) Administer a mixture containing 1 part of kaolin in 4 parts of water by mouth, giving a pint every hour if possible during the acute stage.

(4) Fluids should be restored as follows:

(*a*) Inject intravenously a hypertonic saline solution containing 13.75 grams of sodium chloride and 0.25 gram calcium chloride per liter of distilled water; it should be administered slowly and continuously, if possible, gaging the amount on an estimate of the specific gravity of the blood (normal, 1.056 to 1.058) as follows[6]:

[6] To determine the specific gravity of the blood, prepare a series of solutions of glycerin and distilled water of specific gravities 0.002 apart, from 1.050 to 1.070 (i. e., 1.050, 1.052, 1.054, etc.). Place small portions (10 to 15 cc) of these solutions in small bottles. Drop one drop of blood into each bottle. The specific gravity of the blood is indicated by the bottle in which the drop of blood neither rises to the top nor sinks to the bottom of the solution.

If the specific gravity is—

1.062, give 1,000 cc.
1.063, give 1,500 cc.
1.064, give 2,000 cc.
1.065, give 2,500 cc.

Repeat the saline injections every 4 hours until specific gravity of blood drops below 1.062. If the patient is dehydrated and equipment for determining the specific gravity of the blood is not available, administer hypertonic saline intravenously, using judgment as to amount.

(*b*) If hypertonic saline solution cannot be prepared, inject slowly 1,000 cc physiologic saline solution intravenously every 4 hours until dehydration is relieved.

(5) To control shock in the stage of collapse, add 50 grams (1⅔ ounces) glucose to each 1,000 cc of saline solution administered. Also give 2 milligrams thiamin hydrochloride, 50 milligrams ascorbic acid, and 15 milligrams nicotinic acid amide. If normal human serum or plasma is available for intravenous use, it should be used as a means of controlling shock, but not as a substitute for other fluids that are essential.

(6) To combat anuria or marked acidosis, inject intravenously a solution containing 5.75 grams sodium chloride and 18.25 grams sodium bicarbonate per liter of distilled water. This solution should not be sterilized by boiling or autoclaving as the temperature reached during these procedures will change the bicarbonate to the caustic carbonate; hence dissolve the sodium chloride in the distilled water and sterilize by boiling, then remove the heat and at once add the sodium bicarbonate, which has been taken directly from the original container with a sterile spatula and weighed in a sterile vessel, and finally cool the solution to body temperature and use at once. This solution should be prepared and administered with great care and the patient observed carefully for signs of tetany or other manifestations of alkalosis.

f. Prevention.—Use sanitary measures similar to those employed for the prevention of other enteric infections. Immunize troops entering endemic areas with a suitable vaccine, and revaccinate annually prior to the cholera season.

58. **Filariasis (Bancroft's type).**—*a. Etiologic agent.*—*Wuchereria bancrofti.*

b. Geographic distribution.—The disease is indigenous in

practically all tropical regions of the world. In the Western Hemisphere it is endemic in Central America, South America (Colombia, Venezuela, French, Dutch and British Guiana, and northern Brazil), and the West Indies. In the United States, it is endemic near Charleston, South Carolina.

c. Transmission.—Adult worms (*W. bancrofti*) live in the lymphatic system of infected human beings, releasing larvae (microfilariae) into the lymph and blood streams. A wide variety of night-biting mosquitoes serve as intermediate hosts. *Culex quinquefasciatus* is a common vector. A period of about 10 days is required for development of microfilariae in mosquitoes, during which time the worms migrate to the mosquito's mouth parts. Man is infected by the introduction of worms into the wound made by the mosquitoes' bites.

d. Specific diagnosis.—After an indefinite, prolonged incubation period symptoms are produced by invasion of the lymphatic vessels, most often those of the spermatic cord and of the groin. The initial acute attack is characterized by lymphangitis, accompanied by the usual local signs and fever, and may persist for several days. These attacks recur, and chronic elephantoid manifestations due to blockage of the lymph channels tend to develop, in the lower extremities and more frequently the scrotum and groin. Hydrocele, lymphatic varicocele, epididymitis, and chyluria also occur. Chyluria may be a prominent symptom early in the course of the disease or after evident involvement of lymph channels by the parasite. The urine is cloudy due to the presence of lipoid substances which are soluble in ether. On standing, a clot forms in the urine and a surface pellicle is usually present. The urine also often contains blood due to rupture of a small vessel into the lymphatic varix which is responsible for chyluria. In these circumstances the night specimens of urine often contain microfilariae. The specific diagnosis is made by identification of the microfilariae in blood films. Nocturnal periodicity of the appearance of microfilariae in the peripheral blood stream is characteristic, with increment in the evening and decrement in the early morning hours.

e. Treatment.—(1) No specific treatment is known.

(2) If acute lymphangitis, which is usually caused by hemolytic streptococci, develops, sulfadiazine is recommended as the drug of choice (par. 43).

(3) Surgical measures may be indicated for elephantiasis.

f. Prevention.—Bites of mosquitoes that may serve as immediate hosts should be eliminated, as far as possible, by—

(1) Measures directed toward the prevention of mosquito biting, such as the screening (18 mesh) of residences or the temporary use of bed nets or repellents, if screening is not possible.

(2) The elimination of mosquito breeding places or the use of mosquito larvicides.

59. Hookworm infection.—*a. Etiologic agent.*—*Necator americanus* is almost exclusively the hookworm of man in the Western Hemisphere. *Ancylostoma duodenale* and *N. americanus* are both prevalent in the Eastern Hemisphere. The dog hookworms, *A. braziliense* and *A. caninum*, infect the skin of man in their larval stage, causing the disease known as "creeping eruption." This infection is acquired usually by lying on sandy soil and beaches that have been contaminated by dog excreta.

b. Geographic distribution.—In the United States hookworm infection occurs in the South from Virginia and Kentucky to eastern Texas. The rural population of some counties in South Carolina, Georgia, Alabama, and Mississippi and in northern Florida still show an incidence of 50 percent or over. Hookworm is prevalent in the tropics wherever the climate is moist.

c. Transmission.—The hookworms affecting man live in the small intestine and suck blood from the mucosa. Eggs are passed in the feces and hatch in the soil. Infective larvae develop in a few days, penetrate the skin of man, and reach the intestine via the lungs, trachea, and esophagus.

d. Specific diagnosis.—A specific diagnosis is made by identifying the worms or ova in the feces. In well-nourished adults fifty or more worms are likely to produce clinical symptoms. The principal symptoms are lassitude, fatigue, weakness, pallor, secondary anemia, slight or moderate eosinophilia, and edema.

e. Treatment.—(1) Give a light meal the night before, preferably free from fat; no pre-treatment purge is necessary unless constipation exists. Give one of the two following drugs in the morning on an empty stomach:

(*a*) In the absence of Ascaris infection, give 3 cc tetrachlorethylene in hard gelatin capsules or in 60 cc (2 ounces) skimmed milk (fatal toxicity has never been reported, but dizziness and drowsiness have occasionally occurred).

(*b*) In the presence of Ascaris infection, give 1 gram (15

grains) hexylresorcinol crystoids (Caprokol) (no toxic symptoms have been reported); this will usually remove all ascaris worms and about 50 percent of the hookworms. It should be followed after 3 days by treatment with tetrachlorethylene to remove the remainder of the hookworms.

(2) Avoid food for 4 hours after treatment, and alcohol for 24 hours before and after treatment. A saline purge should be given the following day, if bowels have not moved since treatment.

(3) Iron should be administered to all hookworm cases showing anemia; give 0.35 gram (5 grains) ferrous sulfate (exsiccated) in capsules by mouth three times a day after meals.

(4) The diet should be rich in iron and vitamins.

(5) Stool examination should be made 1 week after treatment; if eggs are found, treatment should be repeated until cure is obtained.

(6) For the treatment of "creeping eruption" repeated use of one of the following methods may be necessary to obtain cure:

(*a*) Apply a piece of cotton saturated with ethyl acetate to the area just beyond the advancing edge of the skin lesion, cover it with adhesive tape, and allow it to remain for 24 hours.

(*b*) With ethyl chloride spray or carbon dioxide snow (dry ice) freeze for 1 minute an area 1 inch in diameter just beyond the advancing edge of the skin lesion.

f. Prevention.—Provide for the sanitary disposal of excreta, and protect the skin against contact with materials contaminated with hookworm larvae.

60. Leishmaniasis.—*a. Visceral leishmaniasis kala-azar).*—(1) *Etiologic agent.—Leishmania donovani.*

(2) *Geographic distribution.*—The disease is widespread, and occurs in certain Mediterranean countries, South Russia, India, China, Manchuria, Abyssinia, Sudan, northern and eastern Brazil, the Chaco region of Argentina, the Paraguay-Brazil border (Matto Grosso), and northern Bolivia (Yungas).

(3) *Transmission.*—The methods of transmission in nature are not yet known. The available evidence suggests that the disease may be transferred by the bites of infected sand flies of the genus Phlebotomus, or other biting files, including the genus Stomoxys, and possibly through the ingestion of food

or drink contaminated with *L. donovani* or by contact. Dogs and other lower animals are susceptible to infection.

(4) *Specific diagnosis.*—The diagnosis depends on the demonstration of *L. donovani* either in smears or cultures of the peripheral blood, tissue juice from lymph nodes, or liver pulp. Inoculation of hamsters may be of assistance. The symptoms include irregular recurring fever with progressive enlargement of the spleen, irregularly undulating fever with a double peak in each 24 hours, anemia, leukopenia, emaciation, and dysentery or diarrhea.

(5) *Treatment.*—(*a*) Administer one of the two following drugs:

1. Inject a freshly prepared 2 percent solution of stibamine glucoside (Neostam) intravenously on alternate days, giving an initial dose of 2.5 cc and increasing each succeeding dose by 2.5 cc to a maximum dose of 10 cc; continue treatment until twelve to fifteen doses have been given. If toxic symptoms develop, reduce the dose.
2. Inject a freshly prepared 2 percent solution of potassium antimony tartrate, C. P., intravenously on alternate days, giving an initial dose of 2 cc and increasing each succeeding dose by 1 cc to a maximum dose of 5 cc; continue until a total of forty or more doses have been given. If toxic symptoms develop, reduce the dose.
3. The toxic symptoms following the use of antimony compounds include nausea, vomiting, dizziness, and collapse. Coughing immediately after administration is not an alarming symptom.

(*b*) The diet may contain whatever foods the patient can tolerate; it probably is wise to supplement it with polyvitamin capsules.[7]

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[7] Polyvitamin capsule—Item No. 1K61500—

Thiamin hydrochloride	1.0 mgm.
Riboflavin	1.5 mgm.
Nicotinic acid amide	10.0 mgm.
Ascorbic acid	37.5 mgm.
Vitamin A	2,500.0 I. U.
Vitamin D	200.0 I. U.

This is one half the daily requirement for the average adult.

(6) *Criteria of cure.*—Cessation of symptoms and the absence of parasites in fluids obtained by hepatic or splenic puncture (relapse may occur).

b. Old World cutaneous leishmaniasis (Oriental sore).—(1) *Etiologic agent.—Leishmania tropica.*

(2) *Geographic distribution.*—Apparently this disease does not occur in localities where kala-azar is present. It occurs in certain Mediterranean countries, Central Asia, Abyssinia, Sudan, Nigeria, and French Congo.

(3) *Transmission.*—As in the case of kala-azar, sand flies of the genus Phlebotomus are suspected as vectors. As the disease can be transferred by inoculation, the possibility of contact infection must also be considered.

(4) *Specific diagnosis.*—The diagnosis depends on the demonstration of *L. tropica* in stained films or cultures of material aspirated from the indurated zone surrounding the ulcer.

(5) *Treatment.*—If the lesions are not numerous, inject into the edges of the sores 2 cc of a 1 percent solution of berberine sulfate; only one to three such treatments may be required. If the lesions are numerous, the treatment outlined above for kala-azar may be used.

(6) *Prevention.*—Take the necessary precautions to avoid the transfer of infectious materials by insects or contact.

c. American mucocutaneous leishmaniasis (espundia).—(1) *Etiologic agent.—Leishmania brasiliensis.*

(2) *Geographic distribution.*—The disease occurs in Mexico, Central America, and South America (Argentina, Bolivia, Brazil, Colombia, Ecuador, the Guianas, Paraguay, Peru, and Venezuela).

(3) *Transmission.*—The exact method is unknown, but it is suspected that sand flies of the genus Phlebotomus may act as vectors, and also that contact infections may occur. The disease is said to be common in forest regions among collectors of chicle-gum and rubber.

(4) *Specific diagnosis.*—The diagnosis depends on the demonstration of *L. brasiliensis* in cultures or smears of material obtained by puncture of the edge of the initial ulcer or in material from nodules in or ulcerations on the mucous membranes.

(5) *Treatment.*—The treatment of the initial lesions is the same as that outlined for Oriental sore (see above). During

the stage of mucous membrane involvement, use the treatment recommended for kala-azar.

(6) *Prevention.*—Use methods similar to those advocated above for the control of kala-azar and Oriental sore.

61. **Malaria.**—*a. Etiologic agents.*—*Plasmodium vivax*, *P. falciparum*, *P. malariae*, and *P. ovale*.

b. Geographic distribution.—The distribution of malaria is world-wide, but that of the different species of parasites varies. *P. vivax*, which is most widely distributed, occurs between 45° N and 45° S latitude, and is the commonest species found in the temperate zones. *P. falciparum* usually is confined to the tropics and subtropics, and in many regions is the commonest species; it rarely occurs where the average summer temperature is lower than 70° F., or the winter temperature cooler than 48° F. *P. malariae* and *P. ovale* are relatively uncommon and their distribution is irregular.

c. Transmission.—Malaria is transmitted from man to man through the agency of certain species of anopheline mosquitoes, the relative importance of which varies in different regions.

d. Specific diagnosis.—(1) The diagnosis depends on the demonstration of plasmodia in blood smears stained by the Wright or Giemsa method. In each suspected case of malaria, blood smears, preferably both thin and thick, should be examined as soon as possible. If no malaria parasites are found, take additional smears on successive days.

(2) In *P. falciparum* infections estimate the proportion of infected erythrocytes, if possible; the demonstration of these parasites is difficult at times, even in the presence of a severe infection with cerebral symptoms, including coma.

(3) When in endemic areas suspect as *P. falciparum* malaria every case of febrile illness in which coma or medical shock occurs. Headache, fever, and prostration are frequent, and often the only prodromal symptoms of cerebral malaria. This form may simulate acute alcoholism, or the patient may be maniacal, requiring morphine. During the stage of onset the temperature is often little elevated, and *in the presence of coma it may be normal or subnormal.* If facilities for immediate examination of blood smears are not available for confirmation of the diagnosis in such emergency cases, immediate therapy should be initiated without awaiting the results of blood smears.

(4) Trauma, surgery, and childbirth in persons with previous clinically latent infections frequently precipitate serious recurrences of malaria. Under these circumstances in endemic regions, malaria should be suspected if fever occurs. All such cases occurring under circumstances in which blood examination cannot be made should receive immediate treatment.

(5) Choice between the methods described below must be based on the particular circumstances and the condition of the patient.

e. Treatment.—(1) Keep the patient in bed.

(2) For field forces, use one of the following methods:

(*a*) *Oral therapy.*

1. Give 0.6 to 1.0 gram (10 to 15 grains) quinine sulfate in capsules or as friable tablets, three times daily before meals for 7 days; 0.02 gram (⅓ grain) plasmochin may be given once daily after food *while* the patient is taking quinine, providing it is administered under the supervision of a medical officer.

2. Give 0.1 gram (1½ grains) atabrine three times daily after meals for 5 to 7 days; 0.02 gram (⅓ grain) plasmochin may be given once daily after food for 3 days *after* completing the course of atabrine, providing it is administered under the supervision of a medical officer.

(*b*) *Emergency intramuscular therapy* (if oral administration is impracticable).—Inject 1.5 grams (22½ grains) quinine hydrochloride or dihydrochloride or 0.2 gram (3 grains) atabrine dihydrochloride intramuscularly, equally distributed between both buttocks, being sure to avoid regions with large vessels and nerves; the site of injection must be thoroughly massaged. The dose may be repeated after 12 hours.

(3) For hospital patients, use one of the following methods:

(*a*) *Oral therapy.*—The Sinton treatment may be used for hospitalized patients. The following mixtures cover one course of treatment for a patient weighing 150 pounds.

Mixture A

Sodium bicarbonate	92.0 grams	(1,380 grains)
Sodium citrate	60.0 grams	(900 grains)
Calcium carbonate	4.6 grams	(69 grains)
Water, to make	690.0 cc	(23 ounces)

Mixture Q

Quinine sulfate	14.0 grams	(210 grains)
Citric acid	42.0 grams	(630 grains)
Magnesium sulfate	84.0 grams	(1,260 grains)
Water, to make	630.0 cc	(21 ounces)

The amount of quinine is calculated at the rate of 0.65 gram (10 grains) daily for each 50 pounds of body weight. Consequently the total varies from 14.0 to 18.2 grams (210 to 280 grains) for patients weighing 150 to 200 pounds. Give a preliminary purge of 0.2 gram (3 grains) calomel, followed by 30 grams (1 ounce) magnesium sulfate dissolved in warm water. After the purge has acted, give 30 cc (1 ounce) doses of mixture A at hourly intervals. One-half hour after the third dose of mixture A, give 30 cc (1 ounce) of mixture Q. Give a 30 cc (1 ounce) dose of mixture A, followed in half an hour by a 30 cc (1 ounce) dose of mixture Q three times daily for 7 days. In addition give 0.02 gram (⅓ grain) plasmochin once daily after food for 7 days.

(*b*) *Parenteral therapy.*—Even though vomiting has not occurred, certain cases receiving oral therapy may fail to absorb the drugs. Examination of the urine by the Binz or Andre method will give evidence as to whether or not absorption is taking place. All such patients, comatose patients, those who cannot swallow or retain medication by mouth, and those with *P. falciparum* infection in whom 5 percent or more of the erythrocytes are infected should be treated intravenously or intramuscularly by one of the following methods until clinical response is obtained or the drugs can be taken by mouth or both:

1. Inject slowly (15 to 30 minutes) 0.3 to 0.5 gram (5 to 7½ grains) quinine hydrochloride or dihydrochloride in 150 to 250 cc sterile physiologic saline solution, containing 0.5 to 1.0 cc of a 1:1000 solution of epinephrine hydrochloride, intravenously every 8 hours. Intravenous fluids must be limited in the stage of coma or collapse, unless the patient is markedly dehydrated. *Injections should not be continued after the patient can take medication by mouth.*
2. Inject slowly 0.2 gram (3 grains) atabrine dihydro-

chloride in 200 cc sterile physiologic saline solution intravenously; do not give more than 0.4 gram (6 grains) in 24 hours. *Injections should not be continued after the patient can take medication by mouth.*

3. Inject 1.5 grams (22.5 grains) quinine hydrochloride or dihydrochloride in 5 cc sterile physiologic saline solution intramuscularly, equally distributed between both buttocks, being sure to avoid regions with large vessels and nerves; this dose may be repeated after 12 hours. *Injections should not be continued after the patient can take medication by mouth.*

4. As soon as oral medication is possible, give quinine or atabrine by mouth (see above).

f. Prevention.—(1) The prevention of malaria may be accomplished either by the elimination of the vectors, by the protection of man from the bites of infected mosquitoes, or by the destruction of the plasmodia in man in order to prevent infection of the mosquito.

(2) The elimination of mosquito vectors is an important factor in the sanitation of permanent posts and camps, but obviously has a limited application in temporary camps occupied by troops on field service. Troops in the field are also unable to avoid areas inhabited by civilian carriers of malaria, who serve as reservoirs for the infection of the local anopheline vectors. Under such circumstances the prevention of malaria must depend mainly on procedures that protect the troops from the bites of infected mosquitoes.

(3) Whenever practicable, camp sites should be on elevated ground at least a mile from swampy or marshy areas and an equal distance from local population groups. The troops should be provided with bed nets made of 20-mesh bobbinet, and medical officers should instruct the personnel as to the use of nets to prevent the exposure of sleepers.

(4) The use of quinine or atabrine for prophylaxis is not recommended as a routine procedure, since the available information indicates that these drugs do not prevent infection. They are, however, of definite military value in that they do prevent the appearance of the clinical symptoms of malaria so long as they are taken, and thus afford a means for keeping troops fit during periods of emergency in the field. When ad-

ministered to troops in special situations in unsanitated endemic areas, either of the drugs may be used under the personal supervision of a responsible officer, as follows:

(*a*) Give 0.2 gram (3 grains) atabrine twice a week (every 3 or 4 days).

(*b*) Give 0.3 gram (5 grains) quinine sulfate daily.

62. Blackwater fever.—*a. Predisposing causes.*—Estivo-autumnal (malignant tertian) malaria and a previous attack of blackwater fever.

b. Precipitating factors.—Chilling, trauma, alcoholism, fatigue, and quinine.

c. Diagnosis.—The three cardinal symptoms are hemoglobinuria, fever, and icterus. At onset the temperature may reach 102 to 103° F. and the patient usually has a chill. Other symptoms are bile-stained vomiting, which may be persistent, abdominal pain, jaundice, or subicteric sclerae, enlarged tender liver and spleen, severe prostration, progressively severe anemia, and elevated plasma bilirubin, with an increased indirect van den Bergh reaction, which may become direct later. There is usually some degree of urinary suppression. The urine varies from light red to black (hemoglobinuria). Asexual forms of *Plasmodium falciparum* are usually absent from the peripheral blood within 2 or 3 hours of onset; crescents and forms of tertian and quartan varieties may be present.

d. Dangerous complications.—These include urinary suppression due to precipitation of acid hematin in the renal tubules, syncope, cardiac failure, hyperpyrexia, uncontrollable vomiting, hiccough, grave anemia, sudden drop in temperature with prostration, and coma.

e. Treatment.—(1) Do *not* give quinine or atabrine until convalescence from the attack of blackwater fever is established.

(2) Provide absolute rest in bed, and keep the patient warm.

(3) Give a minimum of 2,000 cc of fluids per day, much more if possible.

(4) If the urine is acid or anuria exists during the period of vomiting, inject 1,000 cc of physiologic saline solution or of 5 percent glucose solution intravenously; this can be repeated as indicated.

(5) When vomiting is controlled, give 0.6 gram (10 grains) sodium bicarbonate by mouth every 1 to 2 hours until the urine is alkaline to litmus; give it thereafter only if the urine becomes acid.

(6) If the patient is unable to void, catheterize every 4 hours to determine the urinary output and its reaction to litmus.

(7) For severe anemia, give transfusions and repeat daily, if needed.

(8) If plasmodia are present in the blood after convalescence is established, give 0.1 gram (1½ grains) atabrine three times daily for 5 days; watch for the recurrence of hemoglobinuria, since atabrine has occasionally precipitated an attack.

(9) For marked acidosis or anuria (par. 57*e*(6)).

f. Prevention.—(1) Treat every case of estivo-autumnal malaria to complete cure.

(2) Since recurrence of blackwater fever is common, especially in the tropics, send the patient to temperate zone, if possible.

63. Onchocerciasis.—*a. Etiologic agent.—Onchocerca volvulus* (*O. caecutiens*).

b. Geographic distribution.—The disease occurs in Africa and the Western Hemisphere (the western slope of Guatemala at altitudes of 2,000 to 6,000 feet and southern Mexico in the states of Chiapas, Oaxaca, Guerrero, and Yucatan).

c. Transmission.—The adult worms locate principally in subcutaneous tumors in the occipital and temporofrontal regions, releasing microfilariae into the adjacent lymphatics and other tissues. The embryos are found rarely in the peripheral blood. Various species of black gnats (*Simulium*) serve as intermediate hosts. A minimum period of 6 days is required for larval development in the insect, during which time the worms migrate to the gnat's mouth parts. Man is infected by the introduction of the parasite into the wound made by an infected insect.

d. Symptoms.—(1) Tender subcutaneous nodules, ¼ to 1 inch in diameter, are found in the scalp; they are freely movable and easily enucleated.

(2) In about 5 percent of the cases, ocular complications follow migration of microfilariae into the eye, producing keratitis, iritis, and conjunctivitis. Photophobia, xerosis, and impairment of vision follow; sometimes to the extent of complete blindness.

(3) Generalized lichenoid dermatitis with intense itching, particularly at night, may occur.

e. Diagnosis.—A diagnosis is made by the identification of adult worms removed from tumors or of microfilariae from

adjacent tissues (biopsy or aspiration).

f. Treatment.—Treatment consists of the early enucleation of nodules and careful observation for the development of new ones; puncture and aspiration with a hypodermic needle and examination for microfilariae should be performed as soon as a nodule is suspected.

g. Prevention.—(1) The transmitting gnats breed in running streams, the larvae and pupae being attached to rocks or plants beneath the surface; no suitable method of control has been discovered.

(2) Wear flyproof clothing and a veil.

(3) Apply an insect repellent to exposed parts several times a day.

(4) Employ a smudge of smoke to keep gnats away from an encampment or out of buildings.

64. Oroya fever.—*a. Etiologic agent.—Bartonella bacilliformis.*

b. Geographic distribution.—Endemic areas are confined to the hot, narrow valleys of Chile, Peru, Bolivia, Ecuador, and Colombia north of 16° latitude, at altitudes of 3,000 to 10,000 feet.

c. Transmission.—The causative agent is probably transmitted by sand flies of the genus Phlebotomus, principally at night, from January to April. Native dogs with verruga peruana may be an animal reservoir of infection.

d. Symptoms.—(1) In the early, acute stage (Oroya fever)—the incubation period is about three weeks—there is an insidious prodromal period with malaise and low fever, followed by severe illness with a high, irregular fever; severe anemia develops rapidly, and there is severe pain over the long bones. Death occurs in 10 to 40 percent of the cases in the second or third week.

(2) The delayed, chronic stage (verruga peruana) follows the attack of Oroya fever by 30 to 60 days. Initially there are severe rheumatic pains, generally with fever. A miliary (lenticulate or nodular, often granulomatous) eruption appears on the face, trunk, and extensor surface of the extremities, which closely resembles yaws. The eruption may develop on all mucous surfaces and produce severe hemorrhages. The nodules may become gangrenous or bleed profusely. There is no mortality.

e. Diagnosis.—(1) The diagnosis of Oroya fever is established by obtaining positive blood cultures (Battistini's method) or by identification of the organism in stained blood films.

(2) Verruga peruana is diagnosed by a characteristic history of onset and by the presence of typical lesions.

f. Treatment.—There is no specific treatment. Vitamin supplements should be administered, and iron should be used during convalescence.

g. Prevention.—Endemic areas should be avoided especially at night; sand flies of the genus Phlebotomus can penetrate 18-mesh screen.

65. Pinta (carate).—*a. Etiologic agent.*—Various fungi have been suspected, but since 1938 a spirochete, *Treponema carateum*, morphologically identical with *T. pallidum*, has been accepted by some authorities as the causative agent. It may be found in tissue juice from skin lesions and lymph nodes.

b. Geographic distribution.—The disease occurs in Mexico, the West Indies, Central America, and South America.

c. Transmission.—It is prevalent in damp, low-lying tropical regions, affects the dark races chiefly, and is rare in Whites, occurring most often in young adults, from 15 to 25. There is no proof of hereditary transmission, and it is apparently not contagious by patient-to-patient contact. It is probably transmitted by the bite of some blood-sucking insect or other animal parasite (a species of Simulium in some cases).

d. Diagnosis.—The distribution of the skin lesions is localized at the onset, but may be generalized later. The eruption is often symmetrical, at times strikingly so; in extremely rare cases it is unilateral. It is usually located on the face and extremities, often over bony prominences, such as knuckles and malleoli, but it may affect any part of skin except the scalp. The palms, soles, and genitals are rarely affected. Small patches may be present on mucous membranes. The color of the skin lesions is usually a shade of blue (slaty or leaden), which may be either diffuse or stippled. When the blue pigmentation eventually disappears, partial depigmentation is left, which may go on to complete loss of pigment simulating vitiligo. The course of the disease is extremely chronic.

e. Specific diagnosis.—Demonstration of the causative agent in material from typical lesions establishes the diagnosis. Positive Wassermann reactions are obtained only subsequent

to the development of secondary lesions (about 60 percent), and it is only in the advanced cases with marked pigmentation that the positive reactions approach 100 percent.

f. Treatment.—As for yaws (par. 69), the optimum amount and duration of treatment have not been established.

g. Prevention.—Contact with infective lesions should be avoided, and precautions to prevent insect transmission should be taken.

66. Plague.—*a. Etiologic agent.—Pasteurella pestis.*

b. Geographic distribution.—In man or animals, the disease occurs in the United States (Washington, Oregon, California, Idaho, Nevada, Montana, Wyoming, Utah, Arizona, New Mexico, and Territory of Hawaii), South America (Ecuador, Peru, Brazil, Bolivia, and Argentina), the Azores, Africa (all parts), southern Russia, Asia (all parts), and the East Indies.

c. Transmission.—(1) Bubonic and septicemic plague are transmitted by rodent fleas. The former is characterized by enlargement of the lymph nodes (buboes) draining the areas of fleabite, high fever, and great prostration, and the latter by a rapid, overwhelming infection, in which the lymph nodes may not be enlarged.

(2) Pneumonic plague may develop in bubonic cases, from handling infected rodents or by droplet infection from another pneumonic case. It is characterized by a fulminating course, with fever, prostration, and thin, mucoid bloody sputum.

(3) Rats, rabbits, ground squirrels, marmots, and other wild rodents serve as reservoir hosts.

d. Diagnosis.—(1) A smear of aspirated contents [8] of a bubo or sputum stained with methylene blue shows short bacilli (*Pasteurella pestis*) with bipolar staining and swollen, vacuolated involution forms.

(2) A culture from a bubo, the blood or sputum on nutrient agar or in broth shows characteristic gram negative organisms.

(3) Inoculation of mice, rats, or guinea pigs intraperitoneally or by skin scarification produces death in 24 to 72 hours, with

[8] *All infectious material must be handled with the greatest care.* All animals inoculated for plague diagnosis *must* be free of fleas and other ectoparasites prior to inoculation, and thereafter kept in insect-proof cages in a separate room from other animals. All persons handling smears, cultures, cages, or inoculated animals must wear gowns, rubber gloves, and masks, and must observe the strictest aseptic technic.

characteristic lesions and organisms at autopsy.

e. Treatment.—(1) Morphine and sponging should be prescribed as indicated for restlessness and delirium; force fluids by mouth or parenterally for toxemia.

(2) Two types of therapy may be tried:

(*a*) Antiplague horse serum may be effective in bubonic cases if given early. Give 100 to 250 cc or more, depending on the dose recommended by the manufacturer, intravenously and repeat every 8 to 12 hours until the general symptoms abate, due care being taken that the patient is not sensitive to the serum (see par. 44). Serum is manufactured by the Haffkine Institute, Bombay, and by the National Hygienic Laboratories of Argentina, Brazil, Chile, and Peru.

(*b*) Sulfathiazole has been found effective in mice. Its use in human cases has not yet been reported. If used, give by mouth as follows: initial dose, 4.0 grams (60 grains); subsequent doses, 1.5 grams (22½ grains) every 4 hours day and night until the temperature has been normal for 7 days. In fulminating cases sodium sulfathiazole may be tried intravenously as follows: initial dose, 0.06 gram (1 grain) per kilo as a 5 percent solution in sterile, distilled water, given slowly; subsequent doses, 0.03 gram (½ grain) per kilo every 6 hours; change to oral dosage as soon as possible.

(3) Hot, wet applications to the bubo may hasten localization of the infection. Incision should be avoided or delayed until localization is complete, to avoid blood stream infection.

f. Isolation of patient.—This is imperative.

(1) Place the patient in a separate screened room and allow only attendants to enter.

(2) Burn all waste articles contaminated by discharges.

(3) In pneumonic, or suspected pneumonic cases, attendants must wear hoods with goggles or celluloid eye openings, coveralls, or complete gowns with trousers, and rubber gloves.

(4) Sterilize all equipment in contact with the patient by boiling or autoclaving.

(5) Wash the walls and floor of the room and all furniture with 5 percent solution of cresylic acid after discharge of the case; allow the room to air for 48 hours.

(6) Every precaution should be used in the handling of persons dying of plague in order to prevent spread of the infection.

g. Prevention.—(1) Rat-proof buildings and ships.

(2) Destroy rats by trapping, by poisoning with red squill, or by fumigation with cyanide gas. Cyanide fumigation should be performed only by thoroughly trained personnel.

(3) In ports and other cities in endemic areas, examine rats and their ectoparasites for plague.

(4) Avoid contact with wild rodents and rabbits.

(5) In endemic rural areas about camps, control wild rodents by hunting, poisoning, and gassing, and filling of burrows.[9] Constant vigilance must be exercised in and around camps to prevent harborages and access of rats and other wild rodents to food supplies, special attention being given to the thorough collection and proper disposal of garbage.

(6) If a bubonic case occurs, determine the source of the infection. If the source is in a city or town, trap and examine rats from the focus of the infection outward until no more plague infection is found. Then trap and poison from the periphery to the center, and follow at once with rat-proofing and destruction of rat harborages.

(7) If a pneumonic case occurs, quarantine all contacts under guard, and take temperatures every 12 hours for 7 days. Also quarantine under military or police guard the area where the infection was acquired and where contacts may have occurred, prohibit entrance and exit of inhabitants, and make house-to-house inspection, taking temperatures twice daily until 7 days after last case is discovered, and isolating all persons developing fever, regardless of cause. Guards should be cautioned to avoid close contact with quarantined persons. The inspecting personnel should wear gowns, coveralls, caps, masks, and rubber gloves.

(8) Strictly isolate the plague patients, and carefully protect the attending personnel (see above).

(9) Perform antiplague vaccination of military personnel in localities where human plague is occurring or is likely to occur. Vaccination confers partial protection for about 2 years.

67. Relapsing fever.—*a. Etiologic agents.—Borrelia recurrentis* (louse-borne); and *B. duttoni* and others (tick-borne).

b. Geographic distribution.—(1) Louse-borne relapsing fever occurs in central and eastern Europe, northern Africa, India, Malaya, China, Siberia, Japan, and Mexico.

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[9] Consult U. S. Public Health Service for procedures.

(2) Tick-borne relapsing fever occurs in Africa, Asia, Europe, North America (Canada, United States, and Mexico), Central America, and South America (Columbia and Venezuela).

c. Transmission.—(1) Louse-borne relapsing fever is transmitted from man to man by *Pediculus humanus* (head and clothes louse) and possibly by *Phthirus pubis* (crab louse).

(2) The tick-borne variety is transferred from lower animal reservoir hosts to man by soft ticks of the genus Ornithodorus. The reservoir hosts include many kinds of rodents, monkeys, and possibly bats and other mammals. The ticks breed in native huts, in cabins, and in cottages in the recreational areas of the western United States, in caves in the Colorado River Valley, and in the burrows and nests of wild rodents. They invade human habitations, where they have the feeding habits of bedbugs. Attachment to the host for feeding is usually brief (12 to 15 minutes), but may continue for 1 hour.

d. Diagnosis.—Specific diagnosis depends on the demonstration of the parasites in the blood during the typical fever.

e. Treatment.—Inject 0.6 gram (10 grains) neoarsphenamine intravenously. One dose usually cures, but two or three doses may be necessary. This therapy is most effective at beginning of an attack or of a relapse; it is dangerous at the time of crisis or just after it, since the patient may collapse.

f. Prevention.—(1) To prevent louse-borne relapsing fever, apply measures that will prevent the spread of lice from infected to normal persons.

(2) With the tick-borne variety—

(*a*) Avoid occupation of native huts, cabins, or houses in endemic areas.

(*b*) Avoid entrance into caves inhabited by rodents, bats, or ticks.

(*c*) Avoid soiling the hands with blood of rodents in endemic areas.

(*d*) Construct sleeping quarters for military personnel so as to discourage entrance or nesting of rodents beneath the floors or in the walls, or the hiding of ticks in cracks in the floors or walls.

(*e*) Maintain cleanliness of barracks, tents, mattresses, beds, and other furniture and avoid the accumulation of dust or debris.

(*f*) If relapsing fever occurs in military personnel, and ap-

parently is acquired by tick-bite in barracks, make a careful search for ticks and, after thorough cleaning, spray the walls, floors, beds, mattresses, and other furniture with liquid insecticide; sterilize the bedding by heat.

(*g*) Instruct personnel to examine day and night clothing and bedding for ticks after possible exposure.

68. Schistosomiasis (mansoni).—*a. Etiologic agent.—Schistosma mansoni.*

b. Geographic distribution.—The disease occurs in Africa, South America (Brazil, Dutch Guiana, and Venezuela), Central America (possibly Panama and the Canal Zone), and the West Indies (Puerto Rico, Vieques, and the Lesser Antilles, including St. Lucia, Antigua, St. Kitts, Nevis, Montserrat, Martinique, Guadeloupe, and St. Martin).

c. Transmission.—The adult worms live in the veins of the colon and rectum, and the eggs are discharged in the feces. When deposited in fresh water the egg hatches and the embryo (miracidium) enters a snail of the genus *Planorbis* or *Australorbis*. The snails may be found in fresh water with slightly alkaline reaction, usually containing aquatic vegetation, and occasionally in brackish water. They may inhabit slow-moving streams, irrigation ditches, limestone sinks, reservoirs, and small pools after the flooding of streams. The larvae (cercariae), which emerge from the infected snails, enter the water and may infect man or animals by penetration of the skin. Thus the disease may be contracted while bathing, wading, working, or washing clothes in infected water, and possibly through the use of a polluted public water supply.

d. Diagnosis.—(1) Early diagnosis is suggested by a papular dermatitis at the site of penetration of the cercariae.

(2) After 6 to 8 weeks of infection, fever, giant urticaria, bloody stools, and eosinophilia are usually present, and the identification of the lateral-spined ova in stools establishes the diagnosis; proctoscopic examination may be of assistance.

(3) In late cases, rectal polyps, anal fistulas, prolapse of the rectum blood, and ova in the stools, splenomegaly, and cirrhosis of liver may be present.

e. Treatment.—(1) Inject fuadin (Neoantimosan), a 6.3 percent solution of a trivalent organic antimony compound, intramuscularly, giving 1.5 cc, 3.5 cc, and 5.0 cc on successive days, then 5.0 cc on alternate days to a total of ten doses.

The toxic symptoms include vomiting and joint pains (rare).

(2) If a satisfactory response is not attained with three courses of fuadin, use a freshly prepared 2 percent solution of potassium antimony tartrate, C. P., intravenously on alternate days, giving an initial dose of 2.5 cc and increasing each subsequent dose by 1.25 cc up to 7.5 cc; continue until a total of twelve to fifteen doses have been given. The injections should be made 2 or 3 hours after a light meal, and the patient should lie down for 1 hour after administration. The solution should be injected slowly into the lumen of the vein, to avoid thrombosis; the drug causes necrosis if introduced into the subcutaneous tissues. Toxic symptoms include nausea, vomiting, dizziness, and collapse. Coughing immediately after administration is not an alarming symptom.

(3) Antimony preparations are contraindicated in patients with nephritis, jaundice, or severe liver disease.

(4) The diet should liberally supply all the essential nutrient substances. If evidence of hepatic disease exists, it should be high in carbohydrates and low in meat protein; milk, egg, and cheese are permissible. Its supplementation with adequate vitamins [10] is advised.

(5) The criterion of cure is the cessation of the passage of eggs containing living embryos. If cure is not obtained, repeat the treatment after 1 to 2 months.

f. Prevention.—(1) Prohibit entering or using unpurified fresh water in endemic areas. The apparent absence of snails from such water does not guarantee that the water does not contain cercariae.

(2) In case of accidental or necessary entrance into water suspected of containing cercariae, immediate and complete bathing with soap and pure water may prevent infection.

(3) If it is necessary to provide bathing facilities in a natural body of fresh water in an endemic area, a preliminary survey of the surrounding population should be made to exclude the presence of schistosomiasis. Steep banks and wave action diminish the probability of the presence of snails. In an artificial pool fed from a possibly infected source, copper sulfate in a dilution of 1:200,000, will kill cercariae and snails.

69. Yaws.—*a. Etiologic agent.—Treponema pertenue.*

[10] See note 7 (par. 60*a*(5)(*b*)).

b. Geographic distribution.—The disease is common in the tropics, especially in Africa, Polynesia, the Philippines, and some parts of the Western Hemisphere. It is prevalent in the West Indies (especially in Jamaica, Haiti, Trinidad, Antigua, and other islands of the Leeward group), and in the coastal and valley settlements of Colombia.

c. Transmission.—The disease is transmitted by direct contact with human lesions and by nonbiting flies, which convey infective material.

d. Diagnosis.—(1) The initial granulomatous lesion (mother yaw) usually appears on an exposed part of the body.

(2) Generalized or secondary lesions appear in 2 to 8 weeks. These resemble raspberries (frambesiform) or large warts, but smaller papules or scaly, ringworm-like lesions may appear.

(3) Later, disabling lesions, consisting of hyperkeratoses with fissuring or ulceration of the plantar epithelium, may develop; gummatous lesions, like those of syphilis, may occur late in the course of the disease (gangosa—partial destruction of the nose).

e. Specific diagnosis.—Diagnosis is established by the microscopic demonstration of the causative agent in a dark-field preparation of material from typical lesions; the Wassermann and Kahn tests usually become positive 1 or 2 weeks after the appearance of the initial lesion.

f. Treatment.—(1) Yaws responds to the same therapy commonly used for early syphilis, but good results are usually obtained with much less treatment than in syphilis.

(2) The preferred arsenical is mapharsen. The adult dose for men is 0.06 gram (1 grain), and for women 0.04 gram (⅔ grain); neoarsphenamine is also effective, 0.75 gram (12 grains) for men and 0.6 gram (10 grains) for women. The preferred bismuth preparation is bismuth subsalicylate in oil, in doses of 0.2 gram (3 grains). The dose for children should be reduced according to age and weight.

(3) The following standard course of treatment for yaws is recommended: four weekly injections of napharsen or neoarsphenamine and bismuth subsalicylate given on the same day, followed, without a rest period, by four weekly injections of mapharsen or neoarsphenamine alone, which in turn are followed by eight weekly injections of bismuth subsalicylate

alone. Blood for a serologic test should be taken, if possible, at the time of the eighth and the sixteenth treatments.

(4) The patient should be followed by clinical examinations and serologic tests at monthly intervals for 3 months, and then at intervals of 3 months for 1 year. If a clinical relapse occurs or if the serologic test remains positive for 6 months after treatment has been started, the course of treatment outlined above should be repeated.

g. Prevention.—Avoid contact with infective lesions and take precautions to prevent insect transmission.

70. Yellow fever.—*a. Etiologic agent.*—The virus of yellow fever.

b. Geographic distribution.—Yellow fever has previously occurred repeatedly in severe epidemic form in most of the countries of the Western Hemisphere and in Africa. Within recent years it has been shown to be endemic in extensive jungle areas in tropical South America (Bolivia, Brazil, Colombia, Ecuador, Paraguay, Peru, Venezuela, and possibly the Guianas and elsewhere), and in Africa. The transfer of virus from such jungle areas to rural or urban regions where *Aedes aegypti* breed and where the inhabitants are susceptible to yellow fever results in epidemics.

c. Transmission.—Within recent years it has been shown that there are important differences between the mechanisms of transmission of endemic and epidemic yellow fever:

(1) The endemic disease, which for convenience has been designated "jungle yellow fever," probably is maintained in lower animal or arthropod reservoirs, and it probably is transmitted normally by insect vectors other than *A. aegypti.* In Brazil, for example, *A. scapularis*, *A. fluviatilis*, *A. leucocelaenus*, and *Haemogogus capricornii* have been incriminated as effective vectors, and the last two have been found naturally infected. Other species of Aedes and other genera native to North America, Europe, Africa, and the Orient have also been proved to be efficient vectors in the laboratory.

(2) The epidemic disease, also known as "urban yellow fever" or "rural yellow fever" occurs when the virus is introduced into communities of susceptible individuals where the vector *A. aegypti* is available in sufficient numbers. Any area where this mosquito exists is potentially liable to have epidemics of the disease. The breeding range of *A. aegypti* is

between 40° N and 40° S latitude throughout the Old World and New World.

d. Diagnosis.—The symptoms are typical, and the virus of yellow fever may be identified by specific protection tests in animals.

e. Treatment.—(1) There is no specific treatment.

(2) General therapy is as follows:

(*a*) Place the patient in bed in a room screened with 18 mesh screen, and use a bed net.

(*b*) Give a saline laxative at onset, and thereafter enemas as needed.

(*c*) During the acute stage of severe cases give only citrus fruit juices and alkaline water (3,000 cc tap water containing 15 grams (½ ounce) sodium bicarbonate daily); in mild and moderate cases a light diet high in carbohydrates and low in fats is recommended.

(*d*) For the fever, apply an ice cap or cold compresses to the head, and sponge the body with cool water.

(*e*) If vomiting prevents feeding by mouth, give 1,000 cc of 5 percent glucose in physiologic saline solution intravenously three times daily, also give 2 milligrams thiamin hydrochloride, 50 milligrams ascorbic acid, and 15 milligrams nicotinic acid amide, which may be given either parenterally or in the glucose solution. If hemorrhagic manifestations of the disease are prominent, administration of vitamin K combined with bile salts may be helpful.

(*f*) To relieve vomiting, cracked ice and 0.016 gram (¼ grain) cocaine hydrochloride may be given by mouth and 0.032 gram (½ grain) codeine sulfate by hypodermic injection.

(*g*) When the patient is able to eat, he should receive a diet high in carbohydrates and relatively low in protein; milk, eggs, and cheese are permissible. This diet should be supplemented daily with adequate vitamin intake. This regime should be continued until patient is convalescent. If anemia persists during convalescence, liver therapy may be indicated.

(*h*) The patient's activities should be resumed gradually, and a long period of convalescence should be allowed.

f. Prevention.—(1) All military personnel stationed in tropical regions of the Western Hemisphere or in other areas where yellow fever is endemic or is likely to be introduced should be vaccinated against the disease, and all personnel ordered to such

regions for duty should be vaccinated prior to departure. Vaccination usually confers immunity of several years' duration. The recommendations, as given in Circular Letter No. 9, Office of The Surgeon General, February 12, 1941, are as follows:

(*a*) Inject subcutaneously 0.5 cc *diluted* yellow fever vaccine.[11]

(*b*) The vaccine should be given only to persons who are in good health and who are free from acute disease.

(*c*) Slight febrile reactions in 5 to 7 days occur in 5 percent of the cases.

(*d*) Yellow fever vaccine is to be diluted and injected *only* by medical officers.

(*e*) The simultaneous administration of yellow fever virus and vaccine (smallpox) virus should be avoided; when both are to be used, the former should be given first and the latter at least 5 days later. Yellow fever vaccine may be given simultaneously with triple typhoid vaccine or tetanus toxoid.

(2) Special care must be exercised to prevent the introduction of yellow fever into nonendemic areas, through the transfer of infected mosquitoes and human beings. Special care should be exercised to safeguard airplane transportation by the vaccination of flying personnel, the elimination of mosquitoes in planes, and the examination and observation of unvaccinated passengers for signs of infection. (See United States Public Health Service, Foreign Quarantine Division, Unnumbered Circular, dated June 9, 1937, "Measures to be Observed to Prevent Introduction of Yellow Fever into the United States by Aircraft," and Circular No. 71, January 27, 1941, "Quarantine Inspection and Treatment of Aircraft of the Military Forces of the United States.")

(3) In the face of an epidemic, the following general measures should be observed:

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[11] The yellow fever vaccine now available is prepared in the laboratories of the Rockefeller Institute for Medical Research, New York City. It is supplied in ampules containing the desiccated remnants equivalent to 1 cc or 5 cc of living attenuated virus, which must *always* be kept at a temperature not above 4° C. (40° F.), since exposure for 3 or 4 hours at room temperature renders the virus inactive. Before use, this virus must be suspended in the sterile physiologic saline solution supplied in the outfit (full instructions are inclosed), thus making a 1:10 dilution. ***Diluted vaccine that remains unused after 3 hours should be discarded.***

(*a*) Isolate the patient in room protected with 18-mesh screen for 4 days from the onset of symptoms (period of communicability).

(*b*) Fumigate quarters in which the infection may have occurred.

(*c*) Isolate persons who have probably been exposed in a room protected with 18-mesh screen for 10 days (period of incubation), and inspect daily for the onset of symptoms.

(*d*) Vaccinate immediately against yellow fever all persons in the *A. aegypti* infected area who have not already received vaccine.

(*e*) Arrange for daily inspection of the entire military and civilian population in the exposed area to discover any early cases of yellow fever.

(*f*) Perform an autopsy or fiscerotomy on all cases dying of fever of 10 days' duration or less, both in the military and surrounding civilian population, and examine tissue from liver for histological evidence of yellow fever.

(*g*) Institute a vigorous organized campaign for control of the breeding of *A. aegypti.* It is a domestic mosquito and breeds mostly in clear water in artificial containers, such as rain barrels, roof gutters, tin cans, tire casings, and flower vases. The eggs are resistant to drying. The adults are found most frequently in human habitations. Its flight range is limited to a few hundred yards. Screening of 18 mesh is required to exclude it.

71. Diet in treatment of tropical diseases.—In tropical diseases the same dietary principles apply as in all diseases. If the nature of the patient's condition prevents his consuming a wisely constructed diet, deficiencies should be made good with specific supplementary substances administered in such a manner that they will be absorbed.

a. Acute infectious diseases interfere with the intake of food and increase metabolic activities. The requirement for nutritional elements may thereby be increased. This applies to calories, proteins, minerals, and vitamins. Various members of the vitamin B complex are involved in metabolism. They especially enter into the metabolism of carbohydrates. Deficiency disease due to an inadequate supply of factors of the vitamin B complex may be precipitated by fever, vomiting, diarrhea, or the administration of glucose. Therefore, when par-

enteral injections of glucose are administered, it is desirable to guard against vitamin deficiencies. Each 50 grams of glucose should be accompanied by 2 milligrams thiamin hydrochloride, 50 milligrams ascorbic acid, and 15 milligrams nicotinic acid amide, given separately by mouth or parenterally or in the glucose solution.

b. In chronic disease, when a well balanced diet cannot be administered, supplementary vitamins should be given orally or parenterally. The following daily doses are considered minimal: 2 milligrams thiamin hydrochloride; 3 milligrams riboflavin; 20 milligrams nicotinic acid amide; 75 milligrams ascorbic acid; 5,000 international units vitamin A, and 400 international units vitamin D.[12]

c. The medical officer should watch for early clinical signs of manifest nutritional disease, especially glossitis or other evidence of early pellagra, the ocular symptoms and cheilosis of riboflavin deficiency and the peripheral neuritis of thiamin deficiency.

(1) When pellagra can be recognized, give 50 milligrams nicotinic acid amide orally each hour for 10 hours. Repeat the following day until the active symptoms disappear. For clinical riboflavin deficiency, give 3 milligrams riboflavin three times daily by mouth. For the peripheral neuritis of thiamin deficiency, give at least 10 milligrams thiamin hydrochloride orally each day for 1 week.

(2) Scurvy, if detected, should be treated by the daily oral administration of 200 milligrams ascorbic acid and the free use of fresh vegetables and citrus fruits.

(3) Vitamin K deficiency, manifested by hemorrhagic phenomena in obstructive jaundice, in severe liver damage of any kind, and in purpura neonatorum, or resulting from severe diarrhea or lack of intestinal absorption and poor secretion of bile salts may be treated with vitamin K combined with bile salts.

(4) Protein deficiency is encountered in nephrosis, chronic infectious diseases, poor intestinal absorption, severe diarrhea, and in diets poor in protein. If manifested clinically by edema, or acute loss of blood protein, it is best treated by the intravenous administration of human serum or plasma (par. 15).

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[12] See note 7 (par. 60*a*(5)(*b*)).

SECTION VII

RICKETTSIAL DISEASES

72. General.—This section has been prepared as an aid in the diagnosis and control of certain of the rickettsial diseases. It is to be noted that present knowledge offers no specific therapy for this group, and that the control of these diseases depends entirely on their prevention.

73. Typhus fever.—*a. Etiologic agents.*—There are two epidemiological types of typhus fever determined by two arthropod vectors: epidemic or louse-borne and endemic or rat-flea-borne.

(1) *Epidemic typhus* (synonyms—exanthematous typhus, classical, European or Old World typhus, fleckfieber, ship, jail, or camp fever). This type is caused by *rickettsia prowaseki.*

(2) *Endemic typhus* (synonyms—murine, American or flea typhus, Brill's disease). This type is caused by *rickettsia mooseri.*

b. Geographic distribution.—(1) *Epidemic typhus* is found chiefly in Europe, North Africa, Asia, and in the higher altitudes of Central and South America. It is not present in the United States or insular possessions.

(2) *Endemic typhus* is found in Southern United States, including southern California and seaports of the Atlantic coast and insular possessions of the United States, Mexico, and probably coastal South America. It is widespread over Europe, Asia, and Africa, especially in countries bordering the Mediterranean, and in Malaysia, China, and South Africa. About 4,000 cases are reported annually in the United States.

c. Transmission.—(1) *Epidemic typhus.*—The reservoir of the disease is man, and it is transmitted from man to man by the body louse, *Pediculus vestimenti.* Epidemic typhus is chiefly a disease of winter and spring, and affects impoverished, overcrowded, and dirty peoples. Once established, it spreads rapidly from person to person through the agency of the body louse. The large epidemics of history have invariably followed, or been associated with war, famine, or civil revolution.

(2) *Endemic typhus.*—The reservoir of the disease is the rat and the disease is transmitted to man by the rat-flea, particularly *Xenopsylla cheopis.* Endemic typhus is chiefly present in late summer and fall and cases are associated with contact with rats or on premises maintaining rat harbors, for example, grocery stores, food warehouses, restaurants. The disease does not spread from person to person in absence of body lice and is not necessarily associated with lousiness or poverty.

d. Specific diagnosis.—(1) *Epidemic typhus.*—The incubation period is from 6 to 14 days. The onset is variable, usually sudden with chills, fevers, general pains, and headache. The fever continues for about 2 weeks. A macular eruption appears on the fifth or sixth day and rapidly becomes petechial. Typically, the rash appears on the chest or abdomen first, spreads to back, arms, legs, and in severe cases to palms, soles, and face. Mental symptoms are common in severe cases, especially during the second week. One attack confers immunity which is not always permanent. Agglutination of B. proteus OX–19 (Weil-Felix) is positive in the second week, usually in titers above 1:320. Complement fixation with typhus rickettsiae is positive in the second week.[13] The case fatality rate is 20 to 60 percent.

(2) *Endemic typhus.*—The incubation period is from 6 to 14 days. It is clinically and immunologically identical with epidemic typhus, except the rash may be more sparse and fleeting and the clinical course milder than epidemic typhus. The Weil-Felix is positive in the second week as in epidemic typhus. The case fatality rate is about 3 percent. The chief differential diagnosis is from Rocky Mountain spotted fever. Spotted fever is usually a more severe infection than endemic typhus. The rash appears earlier, usually on the third or fourth day, but occasionally may be delayed. Typically, the rash is first seen on wrists and ankles, later spreading to the body. The macules show more tendency to coalesce in spotted fever. The febrile period is about 3 weeks. The Weil-Felix is also positive in spotted fever, but spotted fever is negative for complement fixation with typhus antigen.

[13] Complement fixation in rickettsial diseases is new. It is performed only by the U. S. Public Health Service at present, either at the National Institute of Health, Bethesda, Md., or at the Rocky Mountain Laboratory, Hamilton, Mont.

e. Treatment.—(1) *Epidemic typhus.*—No specific treatment of proven value has been developed. A hyperimmune serum is under study but is not yet generally available. The new sulfonamide drugs have been tried without benefit. The treatment is entirely symptomatic. Absolute rest in bed with good nursing care should be provided. Fluids should be maintained preferably by mouth, rectum, or hypodermoclysis. Constipation may be relieved by enemas or mild laxatives. Tepid sponges should be employed for the control of fever. Headache may be relieved by aspirin or codeine—morphine may prove necessary.

(2) *Endemic typhus.*—The treatment is the same as for epidemic typhus.

f. Prevention.—(1) *Epidemic typhus.*—Prevention depends on the protection of susceptible individuals against infective lice or their immunization with effective vaccines.

(*a*) *Protection against lice.*[14]

1. Delousing of clothing.—Infected articles of clothing can be deloused by heat or storage.

(a) Dry heat, moist heat, or steam heat may be employed. Exposure to *dry heat* should be for a period of five minutes at a temperature of 135° F. It does not injure leather, metal, or webbing, and does not wrinkle clothing. However, it is difficult to secure adequate penetration of the heat into the clothing. *Moist heat* (hot water) may be employed but has the disadvantage of causing shrinkage of woolen goods and damage to felt and webbing. *Steam heat* is efficient; improvised apparatus can be easily assembled. It causes little shrinkage of woolen goods, but produces wrinkling of clothing and damages leather, felt, and webbing. Permanent delousing stations can best use pressure sterilizers. For emergency stations some form of flowing steam such as that provided by the Serbian barrel is the most practicable.

[14] See "Military Preventive Medicine," Dunham, 3d Edition, Chapter XXII.

(b) Lice die if starved. Safe storage time is 30 days in cold weather and 3 weeks in warm weather.

2. *Delousing of troops.*—This may be accomplished by the use of chemicals and bathing. Acetic acid (vinegar) 10 percent solution, kerosene, or gasoline (pure or 50 percent suspension in water) will loosen the eggs attached to hairs and facilitate their subsequent removal by bathing, brushing, and combing. Clipping of the hair is a valuable adjunct to this treatment. Thorough bathing with soap and water is an indispensable part of the delousing process.

(*b*) *Immunization with vaccine.*—Vaccines are now available but their value in the control of typhus epidemics is still under study.

(*c*) *Care of cases and contacts.*—Isolation in vermin-free rooms should be provided. All lice and louse eggs on the clothing and on the patient should be destroyed. Exposed susceptibles should be deloused and quarantined for 14 days after the lice have been removed. The isolation of contacts is not necessary in the absence of body lice.

(2) *Endemic typhus.*—The prevention of endemic typhus is based on the elimination of rats by rat-proofing, trapping, and poisoning. Special care should be directed to the proper protection and disposal of garbage to prevent access by rats. The use of vaccine in endemic typhus is debatable as the incidence of the disease is too low to warrant general vaccination. This procedure is not to be considered in places where the rat population can be controlled.

74. Rocky Mountain spotted fever.—Synonyms (tick fever, exanthematous tick fever, tick typhus, exanthematous typhus of São Paulo).

a. Etiologic agent.—Rickettsia rickettsii.—Synonym (Dermacentroxenus rickettsii). Closely related diseases: Boutonneuse fever (Mediterranean littoral), Kenya typhus (East Africa), Tobia fever (Colombia), and possibly South African tick fever.

b. Geographic distribution.—The disease has been reported from all states of the Union except Maine, Vermont, New Hampshire, Connecticut, Rhode Island, Michigan, and Wisconsin. It is also present in British Columbia and Alberta and in Brazil. About 1,000 cases are recognized annually in the United States.

c. Transmission.—The disease is presumably present in nature in some species of rodents or other animals. Wood ticks and dog ticks (*Dermacentor andersoni* and *Dermacentor variabilis*) transmit the disease to man during the process of feeding. Cases of spotted fever occur principally during the spring and early summer in the northwestern states. In the eastern and southern states the spotted fever season may extend throughout the summer. Rarely, cases occur as late as December.

d. Diagnosis.—(1) The incubation period is from 2 to 14 days, usually about 1 week. The general features are like those of typhus, but are more severe than usually encountered in the endemic form of typhus. The onset is sudden, with chills, fever, generalized aching, soreness of muscles and joints, headache, and prostration. Chills may be repeated for the first few days. Other symptoms may persist throughout the course. Fever is usually of about 3 weeks' duration and is the continuous type with morning remissions of 1° F. to 3° F. Defervescence usually takes place by rapid lysis. Some evidence of meningismus is not uncommon. Delirium, convulsions, and coma may develop in the severe cases.

(2) A macular or maculo-papular eruption appears usually on the third or fourth day of fever. In rare cases the eruption may be delayed a few days. The macules are rose red in color and become petechial as the disease progresses. Typically, the rash first appears on the ankles, feet, wrists, and hands. It spreads rapidly to the legs, arms, back, chest, abdomen, neck, and face. The rash is often seen on the soft palate and the posterior pharyngeal wall. In moderately severe and severe infections the skin of the whole body may be involved within 48 hours of the first appearance of the rash. In some cases fresh macules may appear each day for the first few days. As the disease progresses, the macules become first petechial and bright in color, later becoming dark red or purplish. Coalescence may occur. This may be followed by necrosis of the skin, especially on the ankles or the elbows. The rash in spotted fever almost always persists until defervescence. In many cases the rash persists as brownish spots for weeks or even months after recovery.

(3) The case fatality rate is practically the same for the various sections of the United States. It varies from 12 per-

cent in the age group 1–14 years to 41.8 percent in the age group 40 years and above. Deaths usually occur before the 14th or 15th day of illness.

e. Differential diagnosis[15] (between endemic typhus and spotted fever, based on typical cases).

	Typhus	Spotted fever
Fever	2 weeks	3 weeks.
Rash	First on body; spreads to extremities.	First on extremities; spreads to body.
	Palms and soles usually free.	Palms and soles usually affected.
	Neck and face rarely involved.	Neck and face commonly involved.
	Usually fades before defervescence.	Usually persists until defervescence.
Pulse	100 or less	100–130.
Leucocytes	Normal limits	10,000 to 18,000.
Weil-Felix	Second week	Second or third week.
OX–19	Positive 1:320 up	Positive 1:320 up.
OX–2	Some cases	Some cases.
OX–K	Negative	Negative.
Complement fixation	Positive with typhus antigen.	Negative with typhus antigen.

f. Treatment.—Treatment is essentially the same as in typhus (par. 73*e*). Good nursing care, avoidance of exertion—mental or physical—maintenance of fluids by mouth preferably, by hypodermoclysis if necessary, and relief of headache by aspirin or codeine. There is no specific treatment of proven value. Convalescent serum, transfusions, immuno-transfusion, intravenous glucose, and various chemotherapeutic substances such as metaphen, sulfanilamide, and sulfapyridine have all been tried without evidence of benefit to the patient. A hyperimmune rabbit serum is under clinical trial at the present time. The evidence against the newer chemicals is such as to warrant definite recommendations against their use. Many physicians who have had extensive experience in the treament of spotted

[15] Absolute differentiation depends on isolation of strain in guinea pigs and immunological comparison with known strains. This should be attempted only by individuals with special training and having available adequate equipment.

fever consider any form of intravenous therapy as likewise contraindicated.

g. Prevention.—The season when adult ticks are active extends from late February through early August. There is some variation in this with the climatic conditions and the section of the country. Ticks disappear in the northwestern states about one month or six weeks earlier than in the eastern states. Some years unusually warm weather early in the year may bring the ticks out of hiding a week or so earlier than usual. The disease is transmitted solely through tick contact. Preventive measures are directed toward eliminating or reducing chances of such contact.

(1) Avoidance of tick-infested areas as much as possible. Common sites for ticks are tall grass, especially along animal runs, bushes, along the edge of wooded areas, and along streams. The danger of tick contact can be reduced by clearing out brush and tall grass and the destruction of rodents around camp sites.

(2) Ticks should be removed from clothing and body at least once a day, best done immediately after coming from areas where ticks may be present or as a routine measure on retiring. Ticks are apt to attach to the skin on the hairy portions of the body or where their progress is stopped by constricting clothing, as the belt.

(3) Ticks are best removed with tweezers. The hands should be washed thoroughly after handling ticks. This is especially necessary after removing engaged ticks from dogs or other animals. The site of attachment of a tick should be painted with iodine after the tick has been removed.

(4) The use of vaccine. The spotted fever vaccine is prepared at the Rocky Mountain Laboratory, Hamilton, Mont. It may be procured from that laboratory, from the National Institute of Health, Bethesda, Md., and from State health officers. The vaccine may be given in two or three doses of one or two cc each, one week apart. The evidence indicates that following the first series of injections, one or two doses should be given at the beginning of subsequent tick seasons to insure maintenance of immunity. The immunity established is not absolute, but is apparently sufficient to protect against death should spotted fever be acquired within a year subsequent to vaccination. The vaccine is probably of no value after an

infecting tick contact. It is valueless in treatment. As a relatively small number of cases occur each year, general vaccination is not recommended.

75. Tsutsugamushi.—*a. Etiologic agent.—Rickettsia nipponica* (also it has been named *R. tsutsugamushi, R. orientalis,* and *R. akamushi*).

b. Geographic distribution.—Japan, Federated Malay States, and probably Sumatra and the Philippine Islands.

c. Transmission.—A reservoir of the disease in small rodents which presumably exists in nature. The disease occurs in summer and fall months in low-lying sections that are subject to river floods. The infection is transmitted to man by the larval form of the mite, *Trombicula akamushi.*

d. Diagnosis.—The incubation period is about one week. The general symptoms are similar to those of typhus and spotted fever. The febrile course lasts from 2 to 3 weeks, ending by rapid lysis. The rash is macular or maculo-papular appearing on the second to seventh day after onset, the common day being the fifth. This exanthem first appears usually on trunk and arms, and rapidly becomes generalized. The most striking diagnostic sign is the development of a local ulcer at the site of the infecting mite bite. The regional lymph nodes become enlarged and tender and later there may be a more general lymph-glandular involvement. Leucopenia is usually present. Agglutinins develop for B. proteus OX–K but not for OX–19 and OX–2. The inoculation of the virus into the anterior chamber of the eye of rabbits gives rise to circum-corneal injection and iritis. The rickettsial bodies can be found in Descemet's membrane. The case fatality rate varies from 40 percent to 55 percent. An immunity is conferred by an attack, although second attacks have been noted.

e. Treatment.—The treatment is symptomatic as in typhus and spotted fever (pars. 73*e* and 74*f*).

f. Prevention.—Avoidance of known infected areas during late summer and fall. Frequent examination of body for mites should such areas be visited.

76. Trench fever.—Synonyms (Wolhynian fever, Five-day fever).

a. Etiologic agent.—Rickettsia quintana.

b. Geographic distribution.—Trench fever appeared during the First World War. At that time the disease became epidemic

in nearly all the armies of Europe and later spread to Mesopotamia. Only a few cases have been recognized since the last war but there is some evidence that the disease has retained an endemic foothold in Russia and Poland.

c. Transmission.—The etiologic agent is transmitted by the body louse. Recovered cases of Trench fever may remain infective to lice for months. After a louse has fed upon an infected person, a period of 7 to 10 days passes before this louse can transmit the infection. *R. quintana* in the dried louse excreta remains virulent for at least four months. It also retains its virulence in the dried urine of patients.

d. Diagnosis.—The incubation period is from 2 to 3 weeks. The onset is sudden, with prostration, severe pain in the muscles and bones, particularly the tibia, the radius, and ulna. Frontal headache may be severe. Congestion of the conjunctivae is of common occurrence. The spleen is enlarged. In 70 to 80 percent of the cases a rash consisting of small macules develops, usually on the first or second day of fever. It is stated that this rash does not become petechial as in typhus and spotted fever. The rash is most pronounced over the lower thorax, abdomen, and back. Sweats are common. The leucocyte count is usually between 10,000 and 12,000. The initial bout of fever lasts about five days. Relapses occur at intervals of 5 or 6 days or even longer. Three to five relapses may occur. Prostration is pronounced and convalescence prolonged. The disease is not fatal but is important on account of lengthened disability. Apparently immunity is not established by one attack. The production of agglutinins for B. proteus X (Weil-Felix reaction) has not been adequately tested.

e. Treatment.—The treatment is entirely symptomatic, as in other rickettsial infections.

f. Prevention.—Delousing measures (par. 73*f*). Since the rickettsia of trench fever is present in the urine and saliva of patients, precautions should be taken to guard attendants against contraction of infection by contact. Care should be exercised in disposing of body discharges. Clothing and bedding should be disinfected with steam at 160 degrees F. or above, or by 2 to 3 percent cresylic acid disinfectant.

77. "Q" fever.[16]—*a. Etiologic agent.—Rickettsia burneti (R. diaporica).*

b. Geographic distribution.—Little is known of the distribution of the disease. It has been recognized in human beings in Australia, Montana, and Washington, D. C. There is some question as to the origin of the Washington cases as they occurred in a laboratory where *R. burneti* was being studied, and the possibility of laboratory infection could not be ruled out. Since this disease has been found in widely separated places and man is apparently highly susceptible, it would seem that cases may be passing unrecognized in other sections.

c. Transmission.—In Australia human cases have been associated with the handling of stock animals both on the farm and in the abattoir. An animal reservoir apparently exists in nature and in the bandicoot (a marsupial). Transmission to man by tick, *Hemaphysalis humerosa*, is suggested. In Montana *R. burneti* has been recovered from ticks found in nature (*Dermacentor andersoni*) and one human case has been reported from that section. There is some indication that two types of the disease may exist, one possibly transmitted to man by the bite of a tick and the second contracted through respiratory channels possibly by dust containing the dried excreta of infected animals or arthropods.

d. Diagnosis.—(1) From the Australian reports the incubation period is not too well established. Ten days to 1 month is suggested. The onset is acute with chills, prostration, headache, and fever. The fever, accompanied by chills and sweats, lasts from a few days to 2 or 3 weeks. The white cells are within normal limits. There is no rash and agglutinins are not produced for any of the strains of proteus X so far examined. These two points set the disease somewhat apart from the other known rickettsioses.

(2) One outbreak has been described among laboratory workers in Washington, D. C. These cases were characterized by a rather sharp onset with chills, headache, and fever. The fever lasted an average of 8 or 9 days. The white cells were

[16] "Q" fever is essentially a research problem at the moment. The close resemblance of the clinical picture to that produced by the "virus" pneumonias remains to be explained and the lack of knowledge of the prevalence, distribution, and method of transmission of the disease demands further study.

within normal limits. Insomnia was a common symptom. Vague chest pains and an unproductive cough were present in a majority of the cases. X-ray examination of the chest in each case showed a soft infiltrative lesion. Serial plates usually showed that these lung lesions began centrally and spread outward. In several of these cases the symptoms were not suggestive of lung involvement, but usually a dull note and a few rales could be elicited by the third or fourth day of the illness. Clinically these cases could not be differentiated from the so-called "virus" or "atypical" pneumonias reported frequently in the medical literature of the past few years.

(3) In the second week agglutinins for *R. burneti* appear in the sera of cases. Complement-fixing antibodies for an antigen prepared with the specific rickettsiae also are present. Protective antibodies can be demonstrated in the sera from recovered cases. The causative agent can be isolated in guinea pigs by the intraperitoneal inoculation of blood taken during the febrile period. The identification of strains thus isolated is made by complement fixation and cross immunity tests carried out with known strains. Convalescence from "Q" fever may be prolonged. The fatality rate is apparently low.

e. Treatment.—The treatment is symptomatic. None of the sulfonamide compounds has been found to be of any benefit.

f. Prevention.—So little is known of the transmission of this disease that nothing of practical value can be suggested for prevention.

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[A. G. 062.11 (11-24-41).]

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